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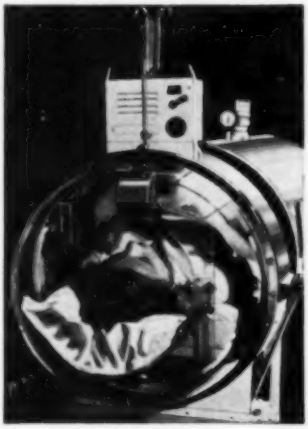

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VOLUME XIX MAY 1951 NUMBER 5

CONTENTS:

COMPLICATIONS FOLLOWING PULMONARY RESECTION OF TUBERCULOSIS IN STREPTOMYCIN TREATED PATIENTS 493
James D. Murphy, M.D. and Harry E. Walkup, M.D., Oteen, North Carolina
Hawley H. Seller, M.D., Orlando, Florida
S. Bornstein, M.D., Oteen, North Carolina

ANGIOCARDIOGRAPHIC FINDINGS IN PULMONARY TUBERCULOSIS 510
Israel Steinberg, M.D., Herbert I. McCoy, M.D. and
Charles T. Dotter, M.D., New York, New York

A CASE OF TUBERCULOUS PERICARDITIS ASSOCIATED WITH A PRIMARY PULMONARY TUBERCULOUS INFECTION 521
Henry C. Sweany, M.D., Jacksonville, Florida

METASTATIC CARCINOMA OF THE PERICARDIUM 528
Frank Lamberta, M.D., Max J. Nareff, M.D. and
Joseph Schwab, M.D., Jamaica, New York

TUBERCULOSIS OF THE GENITO-URINARY TRACT 537
William P. Herbst, M.D., Washington, D. C.

EXPERIENCES WITH DRY BCG VACCINE IN THE PHILIPPINES 544
Sofia Bona-de Santos, M.D., Francisco Duñgo, M.D. and
Alejandro Acanan, M.D., Manila, Philippines

THE ROLE OF PARA-AMINOSALICYLIC ACID (PAS) IN THE EMERGENCE OF STREPTOMYCIN-RESISTANT TUBERCLE BACILLI 566
William G. Lewis, M.D., Laszlo S. Arany, M.D. and
Berkley H. Johnson, M.S., Walla Walla, Washington

PSYCHOGENIC FACTORS IN CHEST DISEASES 570
Arnold S. Anderson, M.D., St. Petersburg, Florida

CLASSIFICATION OF PULMONARY TUBERCULOSIS WITH FIVE DIGIT CODE 577
B. Papanicolaou, M.D., Athens, Greece

STANDARDIZED OBLIQUE ROENTGENOGRAPHY OF THE CHEST 585
Sherman W. Atwell, Brookline, Massachusetts

BENIGN FIBROMA OF MEDIASTINUM 589
J. W. Fouche, M.D., Columbia, South Carolina

MIGRATORY PNEUMONIA WITHOUT EOSINOPHILIA 593
David Biber, M.D., Union, New Jersey
Arthur Buckler, M.D., Irvington, New Jersey

SPONTANEOUS RUPTURE OF THE ESOPHAGUS 598
Henry L. Rigdon, M.D. and N. Douglas Ellis, M.D.
Florence, South Carolina

EDITORIAL: Psychosomatic Medicine 603
C. M. Hendricks, M.D., El Paso, Texas

SEVENTEENTH ANNUAL MEETING 605
American College of Chest Physicians.

COLLEGE CHAPTER NEWS AND NEWS NOTES 614

MEDICAL SERVICE BUREAU xviii

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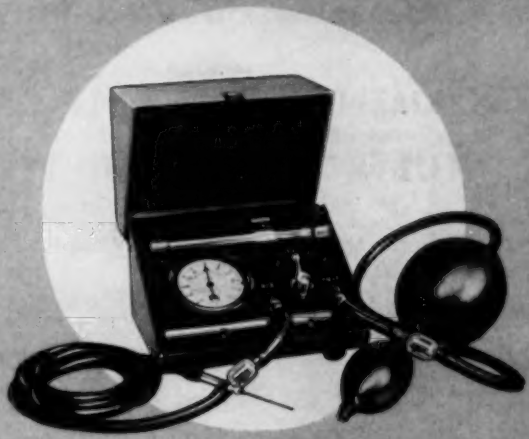

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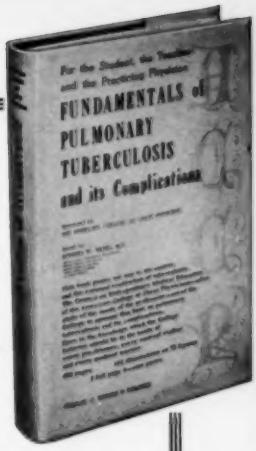
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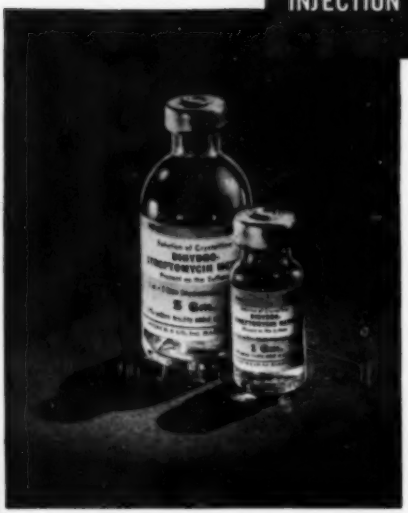

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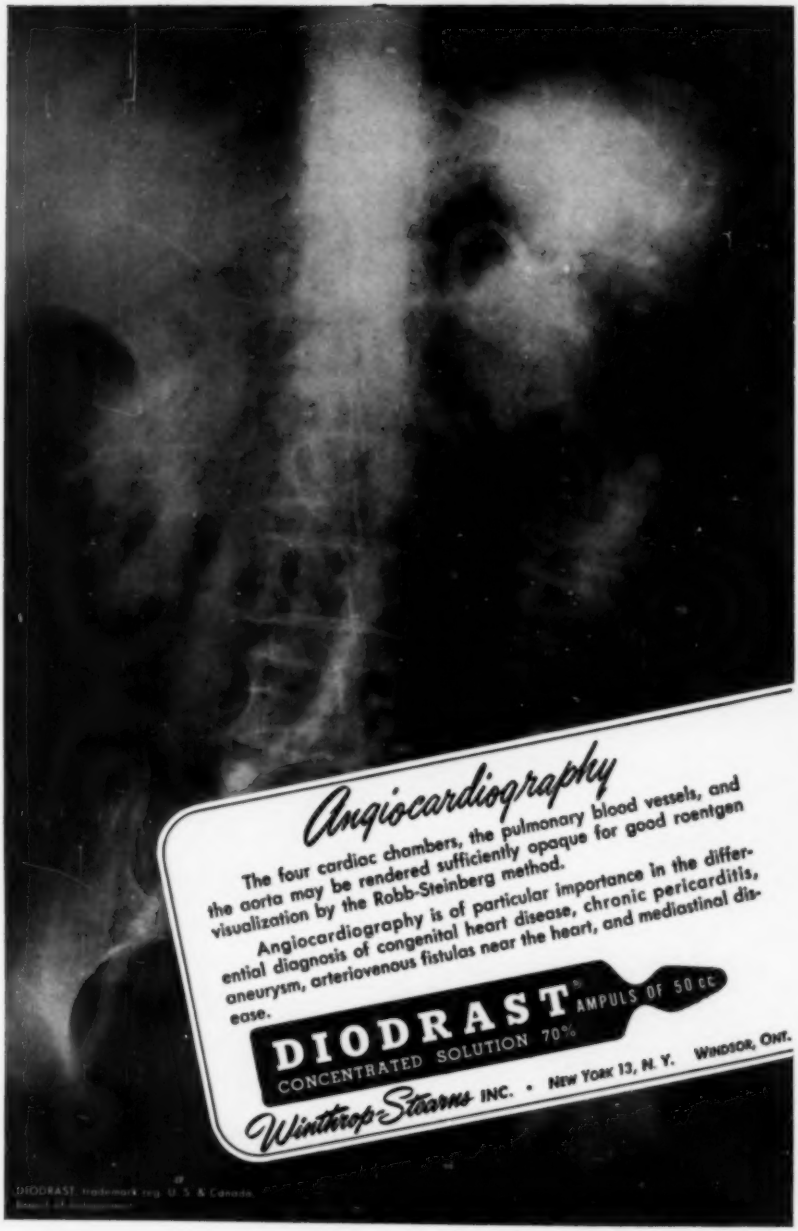
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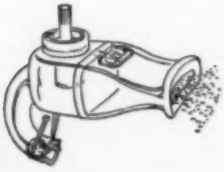

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Complications Following Pulmonary Resection for Tuberculosis in Streptomycin Treated Patients*

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The early results in a series of patients who had pulmonary resection for tuberculosis were reported in May 1948.[1] In this group one to two grams of streptomycin daily were given for a week preceding and two weeks following each resection. The concluding statement in the 1948 report was: "Streptomycin has made it possible for us to perform a lobectomy or a pneumonectomy for tuberculosis with a low morbidity and mortality rate with excellent early results. If late results after pulmonary resection prove to be as gratifying as those after thoracoplasty, we are confident that the indication for pulmonary resection will be greatly extended."

The low mortality rate continues, but with the passage of time, the number of complications, both immediate and late, has increased. This increase has been so marked that our former optimism as to the safety of pulmonary resections protected by streptomycin has diminished. The crux of the problem of resection in pulmonary tuberculosis lies in the ability to obtain a secure and permanent closure of the bronchus. The persistent occurrence of bronchopleural fistulae is evidence that the problem of bronchial closure has not been satisfactorily solved. In the original report 7 per cent of 60 patients developed fistula and empyema. The operative mortality was 5 per cent. At that time there had

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been no late deaths. The present report concerns 146 patients who had pulmonary resections at the Veterans Administration Hospital, Oteen, North Carolina, from January 1947 to May 1, 1950. Sixty-six patients had lobectomy; 80 pneumonectomy. Twenty-two of the total series have developed bronchopleural fistulae. Eight of the fistulae followed lobectomy and 14 pneumonectomy. The per cent of fistulae is now 15 and empyema 14.5.

The diagnosis of fistula was made on an average of 52.7 days postoperatively. Over 60 days elapsed in eight patients before the signs of fistula appeared. There have been only 4.8 per cent operative deaths and 5.5 per cent late deaths in the total series.

This paper will compare the 22 patients who developed bronchopleural fistulae with the entire series of 146 patients. Efforts will be made to gain some knowledge as to the cause for the development of this serious complication. The study will consider:

1) Social and historical data.
2) Indication for the resection.

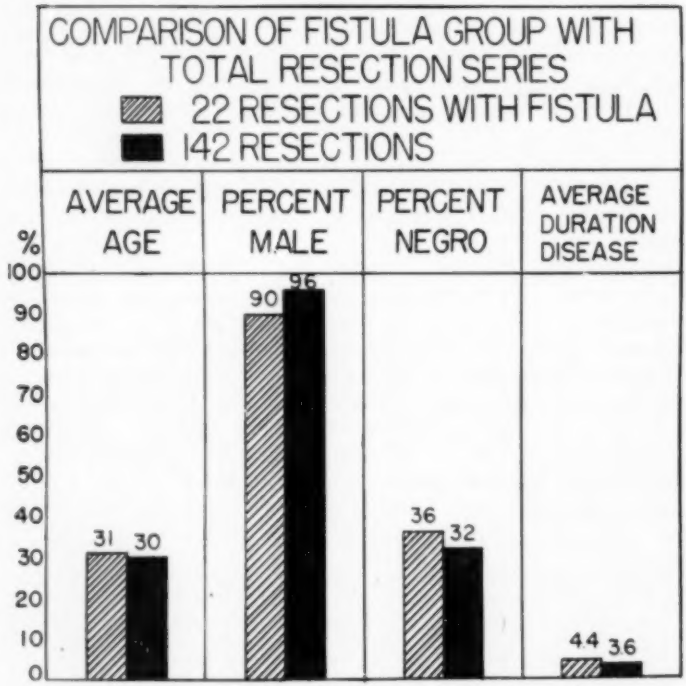

FIGURE 1: Comparison of Fistula Group with Total Resection Series. 22 Resections with Fistula; 142 Resections.

3) The presence of endobronchial involvement preoperatively.
4) The dynamic status of the pulmonary lesion at the time of resection.
5) The character of the disease at the time of resection.
6) Significant pathological details.
7) Significant operative details.
8) The role of streptomycin in prevention of complications.

The first point of comparison between the fistula group and total series of resections concerns itself with social and historical facts. Among these, age, sex, race, and duration of disease prior to resection are the most significant. Figure 1 shows that in all four of these points there is almost an exact similarity between the two groups.

The second major point for comparison lies in the indications for resection in each of the two groups. Of the fistula group 81.7 per cent were done for one of four major reasons: residual cavity after thoracoplasty, failure of conservative collapse measures, destroyed lung, and extensive unilateral disease. Sixty-nine per cent of the total series fell within these four categories. Individually, (Table I) the indications for resection in both groups show little variation except for the destroyed lungs. In this category of indications, 22.7 per cent of the fistula group were placed, whereas, only 8.2 per cent of the total series of resections were done for that reason. This would seem to indicate that resection for such an indication was hazardous. Pleural contamination or decreased

TABLE I
INDICATIONS FOR RESECTION

	Fistula Group		Total Resection Series	
	No.	Per cent	No.	Per cent
Residual cavity	7.0	31.8	55.0	37.8
Failure conservative collapse	3.0	13.6	21.0	14.3
Destroyed lung	5.0	22.7	12.0	8.2
Elective	1.0	4.6	10.0	6.8
Tuberculous bronchiectasis	1.0	4.6	10.0	6.8
Lower lobe cavity	2.0	9.0	6.0	4.2
Extensive unilateral disease	3.0	13.6	13.0	8.9
Bronchiostenosis	—	—	9.0	6.1
Atelectasis	—	—	6.0	4.1
Tuberculoma	—	—	2.0	1.4
Progression after lobectomy	—	—	2.0	1.4
TOTAL	22.0	100.0	146.0	100.0

tissue resistance in this type of patient might be an explanation.

The third point considered was the endobronchial status, as revealed by bronchoscopy, in the two groups prior to resection (Table II). Examination of the table reveals that 18 of the 22 patients, or 81.8 per cent, of the fistula group had no endobronchial disease diagnosed. Of the total series 65 per cent had no endobronchial disease preoperatively. Thirteen and six-tenths per cent of the fistula group and 15 per cent of the total series gave a history of previous endobronchial disease, with treatment, but revealed no active disease at the time of resection. Only one patient of the 22 that developed a fistula, or 4.6 per cent, had active endobronchial disease at the time of resection. Twenty-nine patients, or 20 per cent of the entire series, however, had a diagnosis of preoperative active disease. These figures would indicate that from an endobronchial standpoint those in the group that developed fistulae were in a more favorable condition than the patients in the entire resection series.

The next point of interest was the dynamic status of the pulmonary lesion at the time of resection. We were interested in determining whether the lesion was stationary, progressive or retrogressive immediately before operation. When the two groups were compared, as seen in Table III, six patients, or 27.2 per cent of the fistulae were considered stationary. Seventy, or 48 per cent, of the total series were considered in a stationary status. Thirteen,

TABLE II

ENDOBRONCHIAL STATUS AT TIME OF RESECTION

	Fistula Group		Total Resection Series	
	No.	Per cent	No.	Per cent
No disease	18.0	81.8	95.0	65.0
History of Previous disease	3.0	13.6	22.0	15.0
Active disease	1.0	4.6	29.0	20.0
TOTAL	22.0	100.0	146.0	100.0

TABLE III

STATUS OF PULMONARY LESION AT TIME OF RESECTION

	Fistula Group		Total Resection Series	
	No.	Per cent	No.	Per cent
Stationary	6.0	27.2	70.0	48.0
Progressive	13.0	59.1	61.0	41.8
Retrogressive	3.0	13.7	15.0	10.2
TOTAL	22.0	100.0	146.0	100.0

or 59.1 per cent, of the fistula group were considered to have a progressive lesion. Decision was based upon x-ray evidence and the clinical course, both of which were correlated with the pathological findings. Sixty-one patients, or 41.8 per cent, of the total series had a progressive type of lesion. The retrogressive type of lesion was about equally distributed in both groups. This indicates that patients with progressive type of tuberculosis are more apt to develop bronchopleural fistulae than those with stationary or retrogressive lesions.

The pathological character of the pulmonary disease was considered an important differential point. For classification, the patients were divided among four groups, as revealed in Table IV. The character of the disease process was determined by correlating the pathological reports of the resected specimens with the preoperative x-ray reports. We were interested in determining whether recent or exudative disease had any influence on the subsequent development of fistula. The pathological reports on each resected specimen were reviewed. Whenever the pathologist had described areas of recent exudative disease or pneumonitis that patient was placed in the fibrocavernous exudative group. Those with fibrocavernous disease were considerably more numerous in the total series than in the fistula group. Those with fibrocaseous disease were roughly comparable.

The percentage of patients considered to have fibrocavernous tuberculosis with an exudative or recent component was definitely increased in the fistula group as compared to the total series. Fourteen, or 63.6 per cent, of the fistula series fell with this category, whereas only 41.1 per cent of the entire series had an exudative or recent component. This would indicate that those with an exudative component or a recent pneumonitis offer a worse prognosis as to fistula development than the other classes of patients.

TABLE IV

CHARACTER OF DISEASE CORRELATION OF PATHOLOGICAL AND X-RAY DATA

	Fistula Group		Total Resection Series	
	No.	Per cent	No.	Per cent
Fibro cavernous	4.0	18.2	45.0	30.8
Fibro cavernous-exudative	14.0	63.6	60.0	41.1
Fibro cavernous-caseous	4.0	18.2	39.0	26.7
Tuberculoma	0	0	2.0	1.4
TOTAL	22.0	100.0	146.0	100.0

The relationship of the details of surgical technic and the problems encountered in individual operations were a little more difficult to evaluate. It was decided, finally, to classify them as to: 1) The number and character of pleural adhesions. 2) The presence or absence of pleural contamination during surgery, either from empyema, tearing into the pulmonary cavity during dissection, or from rupture of tuberculous lymph nodes. 3) The relationship of the fistula to the method of bronchial closure.

Several types of bronchial closure were used during the 146 resections. The factors considered of possible etiological significance were: 1) The use of a crushing clamp on the proximal bronchial stump. 2) The use of silk or wire sutures, and 3) The pleuralization of or the failure to cover the bronchial stump following the resection. Figure 2 shows in graphic form a comparison of these several factors in both groups.

The majority of the patients in both the fistula series and the total resection series had what were classed as dense adhesions at the time of surgery. The most striking difference was the

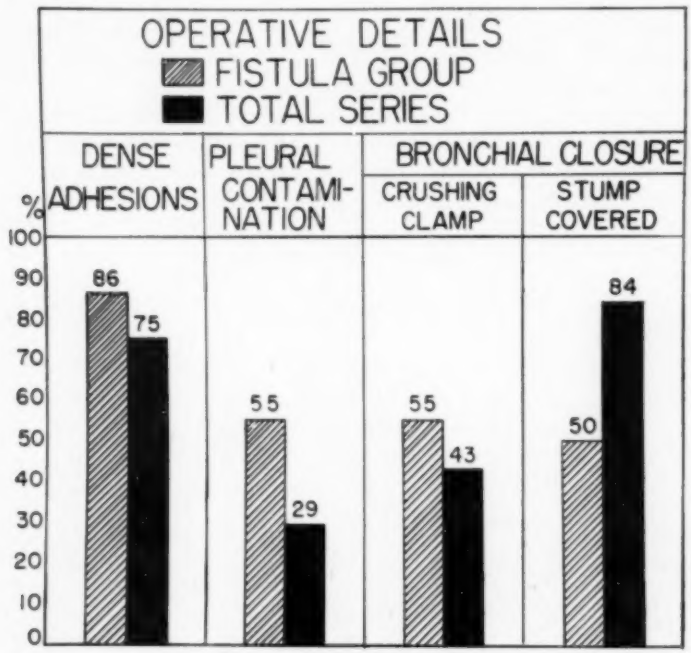

FIGURE 2: Operative Details. Fistula Group; Total Series.

occurrence of pleural contamination in 54.4 per cent of the fistula series as opposed to only 29.4 per cent of the total series. This does appear to be of statistical significance and would indicate that pleural contamination is a more serious calamity than has been commonly considered since the advent of antibiotic therapy.

Bronchial closure appeared to yield similar results whether or not we used a crushing clamp on the proximal bronchus and whether or not we used silk or wire sutures. There was an apparent increase in the rate of occurrence of bronchopleural fistula in those patients who did not have the bronchial stump pleuralized as compared with the total series. In 50 per cent of our 22 fistulae no attempt was made to cover the bronchial stump. In 84.3 per cent of the operations in the total series where the information was available the bronchial stump was pleuralized. This discrepancy, however, is more apparent than real because in 59 of the patients the operative report did not state whether or not there was an attempt made to pleuralize the stump. It is probable that where no definite statement was made as to this factor that the stump was left uncovered and that actually the numbers in each group are about of similar proportions.

Pathology

The absence of gross evidence of bronchial involvement at the line of resection, after examination by the endoscopist, the surgeon, and finally by the pathologist, would make it seem unlikely that such an area would reveal microscopic evidence of involvement. The present study, however, proved that to be a fallacy.

Sections of the bronchial wall at the point of division in 50 patients (26 lobectomies and 24 pneumonectomies) who did not develop fistulae were examined. In 18, tubercles were seen in the bronchial wall. These were usually in the tunica propria and occasionally in or around the bronchial lymph nodes. The tubercles were of early epithelioid type, with or without slight caseation, and with or without lymphocytic wall, but without fibrous capsules. In an occasional instance, evidence of healing was noticeable. The epithelium was intact except for microscopic ulceration.

Similar sections were examined from 21 of the 22 patients who developed bronchopleural fistulae. The specimen was lost in one case. Eight of the 21 patients showed tubercles of the same type, and in the same location as in those of the non-fistula group. Since the per cent of incidence of tubercles at the point of division of the bronchus is almost identical in the two groups, it is obvious that these tubercles are not a significant factor in the development of the fistula. This opinion was recently expressed by Medlar[2] and Auerbach[3] during a discussion of the matter.

Six of the fistula patients have died and autopsies were performed. In five of them, tuberculosis of the bronchial stump up to and including the point of perforation was demonstrated. In the sixth, although it could not be proved that the actual fistula was surrounded by specific granulation tissue, active tuberculosis existed in the immediate vicinity. Thus it appears that the cause of bronchopleural fistulae is an active tuberculous focus that literally eats the stump away.

The question then arises—what is the cause for the infection of the stump? Theoretically three answers are possible: 1) Infection of suture line due to transection of tubercles in the bronchial wall. The equality of occurrence of tubercles in both fistula and non-fistula group makes this hypothesis unlikely. 2) Secretions containing tubercle bacilli collect in the blind sac created by suturing the bronchus and infect the fresh wound. The continued existence of positive sputum after resection would support this theory. 3) Contamination of the pleural cavity at the time of operation results in empyema, which may not be detected until it has eroded through the bronchial stump. That this is the prevalent mechanism is the view recently expressed by Auerbach.[3] In those patients who develop fistulae weeks or months after resection, such a sequence may be involved.

The Role of Streptomycin

Finally, we attempted to determine the part played by streptomycin in the prevention of post-resection complications. From January to October 1947, each patient was given a daily dose of 2 grams of streptomycin intramuscularly for one week before and two weeks after each resection. Since October 1947, the dosage has been one gram daily.

In the early days of the study, the patients who came to surgery had not received streptomycin prior to their resection course. As time passed, and the application of antibiotic therapy became more widespread, an increasing number of patients who were proposed for resection had been given one or more courses of streptomycin prior to their surgery. At present, it is unusual to receive a patient on the surgical service who had not previously had the drug. Many of them have resistant organisms.

Eighteen months ago we began to call attention to the fact that therapeutic results in our hands, expressed in terms of prevention of complications, were apparently inferior in those patients who had one or more courses of streptomycin prior to resection. This inferiority was more apparent when the results in the resistant cases were compared with those in patients with sensitive organisms. A sufficient number of cases have now been accumulated

to confirm this belief. A similar trend was apparent in the large series (630) resections for pulmonary tuberculosis reported by the Ninth Streptomycin Conference in St. Louis in April 1950.[4]

Figure 3 compares the results in our own series of 60 sections (33 lobectomies and 27 pneumonectomies) which were reported in May 1948 with our total series of 146 resections and with 630 resections reported at the Ninth Streptomycin Conference. In the 1948 report, few patients had been given antibiotics prior to resection. The results show that the important complications such as empyema and fistula are twice as common in the total series as in the early group of 60 patients. Spread or exacerbation is nine times as common in the total series.

In Tables V and VI, these results are broken down further, and indicate that pneumonectomy in each group is more productive of complications than lobectomy. In spite of the occurrence of these complications, the operative mortality and late mortality remains at a low figure. The sputum conversion rate in the series of 146 resections is 83.5 per cent. Since some of these operations were performed over three years ago, the overall picture is encouraging.

Our series was broken down further into those who had received no streptomycin prior to their resection and the group who had been treated previously. Eighty-seven patients were in the first group and 52 in the latter. The comparison of these groups is more startling.

Figure 4 shows that in those patients with previous streptomycin

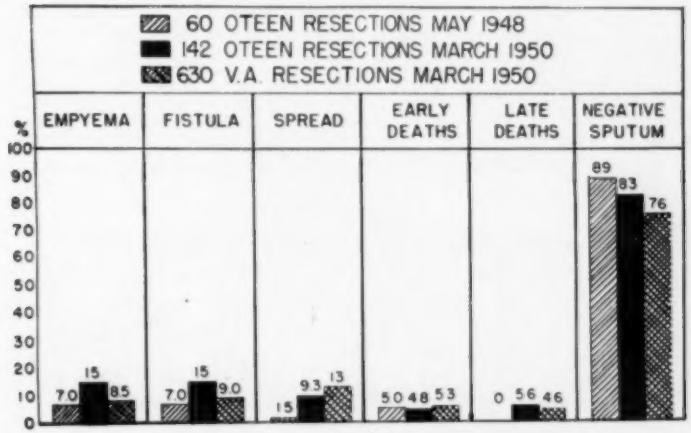

FIGURE 3: 60 Oteen Resections May 1948; 142 Oteen Resections March 1950; 630 Veterans Administration Resections March 1950.

TABLE V

60 RESECTIONS MAY 1948

	No.	Empyema	Fistula	Spread	Deaths E	Deaths L	Negative Sputum
Lobectomy	33	3.0	3.0	3.0	3.0	0	90.0
Pneumonectomy	27	11.0	11.0	0	7.0	0	89.0
TOTAL	60	7.0	7.0	1.5	5.0	0	89.5

Expressed in per cent. E=Early. L=Late.

TABLE VI

142 RESECTIONS MARCH 1950

	No.	Empyema	Fistula	Spread E	Spread L	Deaths E	Deaths L	Negative Sputum
Lobectomy	63	8.0	12.7	4.7	11.1	4.7	4.7	83.6
Pneumonectomy	79	21.5	17.7	5.0	5.0	5.0	6.3	83.5
TOTAL	142	14.7	15.2	4.8	8.0	4.8	5.5	83.5

Expressed in per cent. E=Early. L=Late.

TABLE VII

87 RESECTIONS WITHOUT PREVIOUS STREPTOMYCIN

	No.	Empyema	Fistula	Spread E	Spread L	Deaths E	Deaths L	Negative Sputum
Lobectomy	40	0	0	2.5	15.0	7.5	0	84.2
Pneumonectomy	47	12.7	10.6	6.8	2.3	4.2	0	91.4
TOTAL	87	6.3	5.3	4.6	8.6	5.8	0	87.8

Expressed in per cent. E=Early. L=Late.

TABLE VIII

52 RESECTIONS WITH PREVIOUS STREPTOMYCIN

	No.	Empyema	Fistula	Spread E	Spread L	Deaths E	Deaths L	Negative Sputum
Lobectomy	22	22.6	36.3	9.0	4.5	0	13.6	81.7
Pneumonectomy	30	33.3	30.0	13.1	26.6	6.6	16.6	70.0
TOTAL	52	27.9	33.1	11.0	15.6	3.3	15.1	75.8

Expressed in per cent. E=Early. L=Late.

treatment, empyema was four and a half times, and fistula six times more common than in the previously untreated group. Spread and late deaths were also much higher in the treated group. Negative sputum was obtained in only 75.8 per cent of the previously treated, as compared to 87.8 per cent of the untreated patients.

Tables VII and VIII again break these figures down into lobectomy and pneumonectomy groups. Since the rate of occurrence of complications was higher in the previously treated than in the previously untreated patients, it seemed desirable to determine the relationship of streptomycin resistance to the problem. A study of our own patients revealed that 15 were known to have sensitive and 14 resistant organisms at the time of resection.

Figure 5 illustrates that in this small group of 29 patients bronchopleural fistula occurred twice as frequently in the resistant patients as in the sensitive ones. Empyema—early spread and late deaths followed the same pattern, but not in as marked a degree. Negative sputum was obtained in only 58.3 per cent of the resistant cases, as compared to 80 per cent of the sensitive.

In order to determine whether these results were peculiar to ourselves, or were being experienced by others, we analyzed the streptomycin treated resections reported at the Ninth Streptomycin

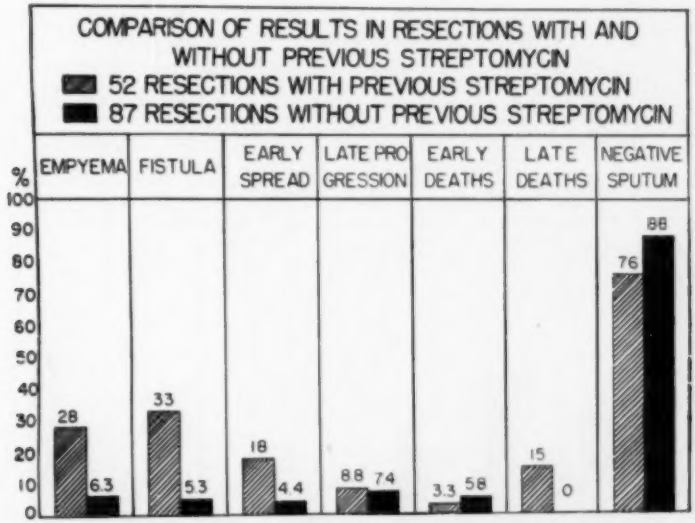

FIGURE 4: Comparison of Results in Resections with and without Previous Streptomycin. 52 Resections with Previous Streptomycin; 87 Resections without Previous Streptomycin.

Conference in St. Louis, April 18-22, 1950. At that meeting, the combined results from 47 Veterans Administration, Army and Navy Hospitals in the United States were presented—630 resections were reported.

In Figure 6, it is seen that 329 resections were performed without previous streptomycin treatment, and 289 with previous treatment. Empyema, fistula, and spread were roughly three times as common in the previously treated, as compared to the previously untreated. Sputum conversion was obtained in only 68.4 per cent of the latter group, as compared to 81.4 per cent of the former.

Figure 7 shows that the same increased prevalence of complications in streptomycin resistant as compared to the streptomycin sensitive group was found among this larger series of 176 patients in whom sensitivity studies were available as was encountered in our small group of 29 patients.

One explanation offered for the increased prevalence of complications is that the protection offered by streptomycin has caused us to operate on patients who would have been rejected earlier. The analysis of our own cases does not completely sustain this viewpoint. Our fistula group was similar in almost every respect to the total series. In age, sex, race, and duration of disease, they were almost identical. There were three points in which the two groups differed markedly. These were the higher ratio of resec-

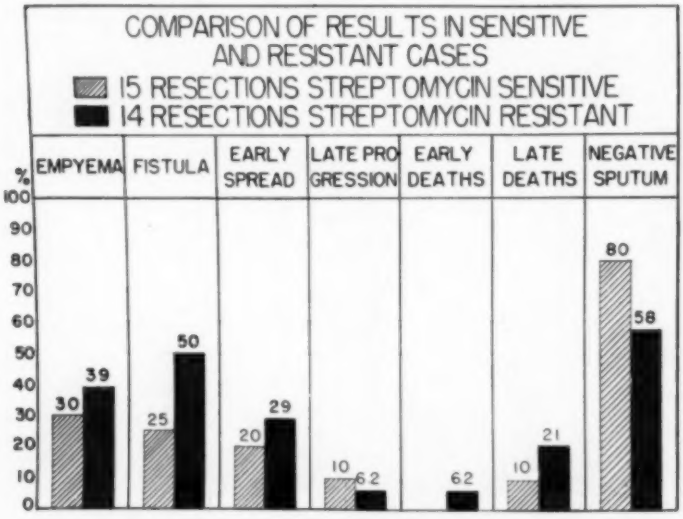

FIGURE 5: Comparison of Results in Sensitive and Resistant Cases. 15 Resections Streptomycin Sensitive; 14 Resections Streptomycin Resistant.

tions done for destroyed lung, the increased number of patients with recent or exudative disease, and the increase in prevalence of pleural contamination in the fistula group. The conclusion is almost unavoidable that previous treatment with streptomycin alters the resistance of the patient in some unexplained manner, so that complications are more common in previously treated patients. This difference is more marked when known resistant are compared with known sensitive cases.

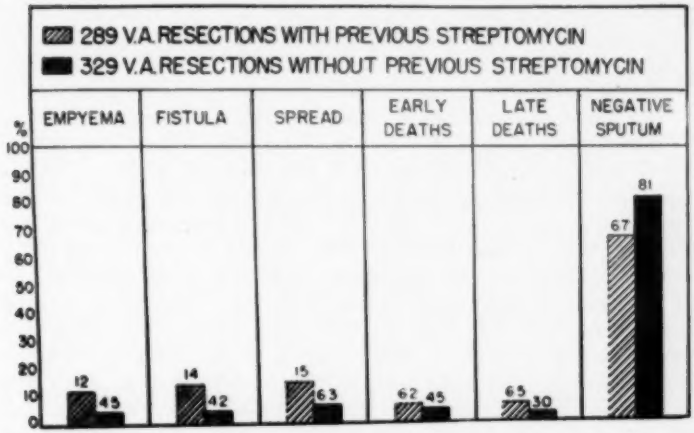

FIGURE 6: 289 Veterans Administration Resections with Previous Streptomycin; 329 Veterans Administration Resections without Previous Streptomycin.

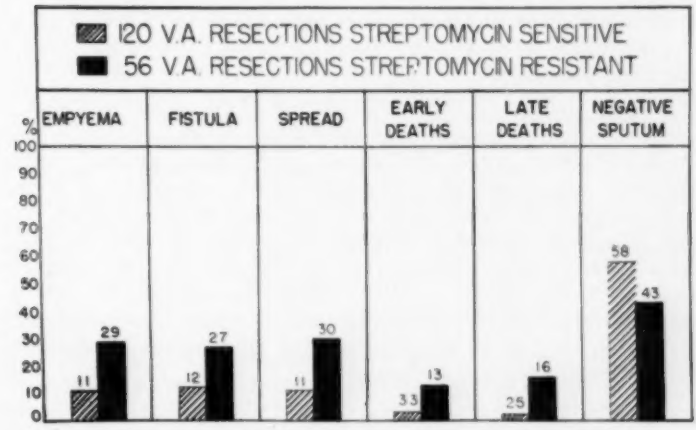

FIGURE 7: 120 Veterans Administration Resections Streptomycin Sensitive; 56 Veterans Administration Resections Streptomycin Resistant.

Discussion

Complications following pulmonary resection for tuberculosis have been more common in the total series of 146 resections than were encountered in an earlier group of 60 patients. The most serious of these complications is bronchopleural fistula which is usually followed or initiated by an empyema. Complications following pneumonectomy are more numerous than after lobectomy but in both procedures they are too high for complacency. Favorable early results had caused us to speculate as to the possibility of resection replacing thoracoplasty as a primary major surgical collapse procedure. We now are convinced that in any situation where thoracoplasty offers a reasonable chance of success it should be the initial step. Resection after thoracoplasty is less of a shock to the patient than when used as a primary procedure. In the prone position and with adequate exposure the surgery itself is also easier. Fistula followed only 11.2 per cent of resections for residual cavities in our series. Seventeen and eight-tenths per cent of patients who had resection done for other reasons developed fistulae. It was considered significant that none of the 39 patients who had resection for residual cavities without having had a previous course of streptomycin treatment developed a fistula, whereas, seven, or 30.4 per cent, of the previously treated group incurred this complication. The resistant patients in this group also proved to be more subject to postoperative fistulae than did the sensitive ones. Sputum conversion has followed 83.8 per cent of resections done for post-thoracoplasty cavity, as opposed to 83.5 per cent in resections for other indications. The occurrence of fistula in post-thoracoplasty resection is not quite as much of a catastrophe as in primary resection since the empyema space has been diminished.

It could be that in the middle portion of our series we encountered the law of averages and overcompensated for some of our more fortunate earlier experiences. After doing 52 lobectomies without a fistula we were disheartened to encounter six in the next 10 cases. The fistulae usually occurred in the patients who had the most surgically satisfactory closure of the bronchus. More recently the pendulum has swung back to the favorable side and in the last 21 resections fistula has occurred only twice. One of these had empyema and fistula before pneumonectomy and the other had an open fistula as a result of an unsuccessful cavernostomy, before lobectomy.

We have compared the fistula and non-fistula group from every standpoint which we thought might influence the development of complications and several points stand out:

1) Resection for destroyed lung is hazardous. Twenty-two and seven-tenths per cent of our fistula group were done for this indication as compared to 8.2 per cent of the total series. It may be that Sarot's procedure of extrapleural pneumonectomy will decrease the operative hazards in such cases.

2) Active endobronchial disease has not proved to be a contributory factor to the development of fistula in our patients.

3) Those with progressive type of tuberculosis with pathological and x-ray evidence of recent or exudative disease have shown a higher proportion of postoperative complications than the total number of patients in our series.

4) Technical operative details such as suture material, type of suture of the bronchial stump, the use or failure to use a pleural cover of the bronchial stump and the use of a clamp on the proximal bronchus have not been determined to influence the number of complications. Pleural contamination, however, has resulted in an increase in complications.

We are convinced that the administration of an earlier course of streptomycin in treatment of pulmonary or extrapulmonary lesion results in an increase in the number of postoperative complications. This increase is marked if the patient's organisms are resistant to streptomycin at the time of resection, but it is also apparent in those previously treated patients whose organisms are sensitive. The experience of the Veterans Administration Thoracic Surgical units, as shown in the reports of the Ninth Streptomycin Conference, also bear this out. Just what the mechanism of this increased susceptibility to complications is has not been determined.

Since the use of streptomycin has become so widespread, it is essential that every patient be studied at the beginning of his treatment program by a cooperative group including both internists and thoracic surgeons. An attempt should be made to determine the possibility of the patient's requiring a resection at a later date and if such a contingency is considered probable streptomycin should be reserved for use during resection, if possible. It must be decided whether the present needs outweigh the benefits to the patient of having the protection afforded by the drug during the surgical phase of his treatment.

SUMMARY

1) An analysis is presented of the case histories of 22 patients who developed bronchopleural fistulae following pulmonary resection for tuberculosis.

2) A similar study was made of our total series of 146 resections which included the 22 fistula patients.

3) A comparison of those two groups indicates that they show no remarkable variation except for the higher rate of development of complications in patients with destroyed lungs and those with a substantially exudative or recent component in the pathological specimen.

4) Previous treatment with streptomycin decreases the protective power of this antibiotic when it is used as an adjunct to a subsequent resection. This diminution of protective power is more marked in patients with resistant than with sensitive organisms. In patients with sensitive organisms, however, the complication rate is higher than in those who are in a virginal state in relation to streptomycin at the time of their resection.

5) Similar experiences with resection in streptomycin protected patients were reported at the Ninth Streptomycin Conference in a series of 630 resections.

RESUMEN

1) Se presenta un análisis de los protocolos de 22 pacientes en los que se desarrollaron fístulas broncopleurales después de resecciones pulmonares por tuberculosis.

2) Se hizo un estudio semejante de nuestra serie total de 146 resecciones, que incluye los 22 pacientes con fístulas.

3) La comparación de los dos grupos no demuestra diferencias notables, con la excepción de la más elevada frecuencia de complicaciones en pacientes con pulmones destruidos y en aquellos con un componente substancialmente exudativo o reciente en el espécimen patológico.

4) Previo tratamiento con estreptomicina disminuye el poder protector de este antibiótico cuando se le usa como adjunto a una resección subsecuente. Esta disminución en el poder protector es más marcado en pacientes con organismos resistentes que en aquellos con organismos sensibles. Sin embargo, en pacientes con organismos sensibles la frecuencia de complicaciones es más alta que en los que están en un estado vírgen, en relación a la estreptomicina, al tiempo de su resección.

5) En la Novena Conferencia de Estreptomicina se comunicaron experiencias semejantes con la resección en pacientes protegidos con estreptomicina, en una serie de 630 resecciones.

RESUME

1) Les auteurs rapportent les observations de 22 malades dont l'exérèse pulmonaire pour tuberculose fut suivie de l'apparition d'une fistule broncho-pleurale.

2) Ils rapportent également l'étude de la totalité de leurs cas,

qui s'élève à 146 exérèses, parmi lesquelles sont compris les 22 cas avec fistule.

3) La comparaison des deux catégories de malades, ceux qui ont eu une fistule, et ceux qui n'en on pas eue, ne montre pas de différences essentielles, sauf que les complications surviennent surtout chez les malades atteints de "poumon détruit" et chez ceux qui ont des altérations anatomiques exsudatives ou récentes.

4) Un traitement préalable à la streptomycine diminue le pouvoir protecteur de l'antibiotique quand il est utilisé pour une exérèse ultérieure. Cette diminution du pouvoir protecteur est plus nette chez les malades dont les bacilles sont résistants que chez où ils sont encore sensibles. Chez les malades dont les bacilles sont encore sensibles, le taux des complications est cependant plus élevé que chez ceux qui n'ont jamais eu de streptomycine avant l'intervention.

5) De semblables constatations chez les malades opérés alors qu'ils avaient été traités par la streptomycine ont été rapportées à la Neuvième Conférence de la Streptomycine en s'appuyant sur 630 exérèses.

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Angiocardiographic Findings in Pulmonary Tuberculosis*

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Earlier reports have recorded preliminary angiocardiographic studies in pulmonary tuberculosis.[1-3] The purpose of this paper is to summarize the angiocardiographic experience in patients with pulmonary, hilar and mediastinal tuberculosis studied over a 14 year period. Knowledge of the status of the vascular supply to the lung affected by tuberculosis is of considerable importance, for the efficiency of the vascular supply bears a direct relationship to the efficiency of respiration. The effect of alteration in pulmonary circulation on the healing of tuberculosis may be significant as the tubercle bacillus has been shown to be an obligate aerobe requiring comparatively large amounts of oxygen for life and growth.[4,5] It is conceivable that the relative hypoxia resulting from local diminution in circulation could have an adverse effect upon the tubercle bacillus.[6] Diminution in circulation occurring in association with tuberculous lesions has been reported in post mortem pathological studies.[7-11] These changes have been visualized roentgenographically by the injection of radiopaque substances in autopsy specimens.[12,13] In the living, Carvalho and his co-workers utilized the cardiac catheter to inject a concentrated solution of sodium iodide. He was able to visualize a diminution in pulmonary vascularity associated with tuberculosis.[14] Angiocardiography is a more practical method of visualization of the circulatory changes in tuberculosis.

Method

For the most part angiocardiography was conducted as originally described using a standard stereo-cassette changer permitting two films per injection.[15] Contrast substance rapidly injected into an antecubital vein passes into the right heart chambers, then to the pulmonary arteries returning via the pulmonary veins into the left heart chambers, finally opacifying the aorta. Roentgenograms were taken at appropriate intervals to best visualize the heart, great blood vessels and lesser circulation.

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In some cases, an automatic roll-film magazine was used enabling roentgen exposures to be made at half-second intervals during the period of opacification. Either Neo-Iopax 75 per cent or Diodrast 70 per cent were used as the contrast substance. Neither compound has resulted in activation of tuberculosis or iodism since the iodine is so bound in the organic molecule as to be unavailable to the body.

Ninety-one patients with pulmonary, hilar and mediastinal tuberculosis have been studied angiocardiographically. These cases have been classified into groups according to the duration of the disease, the clinical and laboratory findings, the roentgen appearance of the tuberculous lesions and in some, post mortem examination as follows: fibro-cavernous, 49; caseo-cavernous, nine; caseous, eight; fibro-calcific, seven; fibroid, six; hilar and mediastinal lymph node tuberculosis, six; hematogenous (non-miliary), three; miliary, one; and silico-tuberculous, one. Of these 19 were classified as minimal, 46 as moderately advanced and 26 as far advanced pulmonary tuberculosis. Of the total number, at least 39 patients received collapse therapy of one form or another and are included in special reports dealing specifically with the angiocardiographic changes following artificial pneumothorax,[16] thoracoplasty, artificial pneumoperitoneum and phrenic nerve crush.[17]

Interpretation of pulmonary vascular changes was made from comparison of the opacified pulmonary circulation of the diseased and non-diseased portions of the lungs as well as from comparison of serial contrast studies in the same individual. These changes included variations in caliber, degree and rate of filling, position and configuration of the pulmonary arterial tree.

FIGURE 1 (T.C., No. 310): Fibro-cavernous tuberculosis of the right upper lobe in a 62 year old white man who also had hypertensive cardiovascular heart disease.—A: Angiocardiogram shows absence of opacification of the right ascending branch of the pulmonary artery with avascularity of the right upper lobe.—B: Tracing of A.

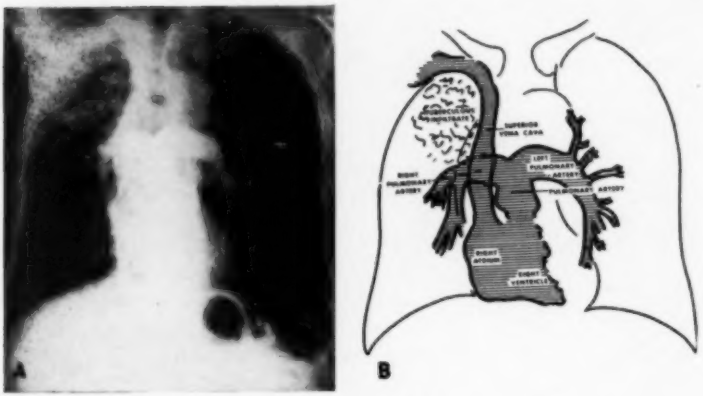

FIGURE 2 (G.G., No. 523): Pericardial effusion in a 22 year old white woman with hematogenous tuberculosis.—A: Angiocardiogram shows opacification of the right atrium, right ventricle and beginning opacification of the pulmonary artery. Pericardial fluid is demonstrated beyond the right atrium.—B: Tracing of A.

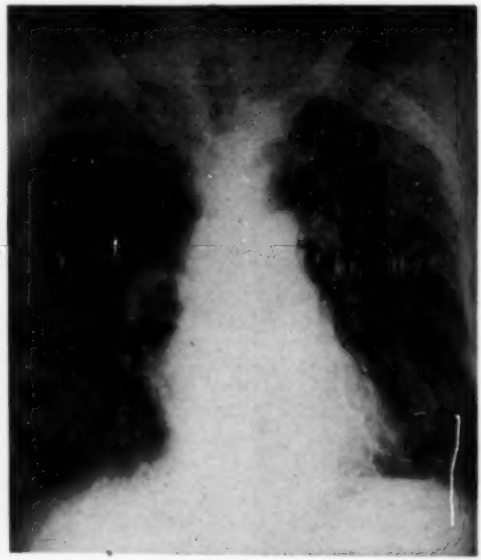

FIGURE 3 (M.K., No. 234): Fibro-cavernous tuberculosis in a 40 year old white man. Angiocardiogram shows abrupt termination in a huge cavity of the apical posterior branch of the left ascending pulmonary artery. The right upper lobe also shows diminished vascularity.

Findings

Diminished vascularity of varying degree occurs in local areas of the lung involved by tuberculosis. This may be seen angiocardiographically by reduced opacification of vessels as illustrated in the ascending branch of the right pulmonary artery in Figure 1 or by absence of opacification, as in the corresponding pulmonary arterial division in Figure 2. Figure 3 shows avascularity of the right upper lobe, while on the left side the posterior-apical segmental pulmonary artery ends abruptly at the site of a large left apical cavity. Marked diminution in caliber and filling of the pulmonary arterial vessels of the right lower lobe is demonstrated in Figure 4. The diminution in vascularity seen in Figure 5 involves both upper lobes. In each instance the decrease in pulmonary vascularity is found to be intimately associated with the tuberculous lesion. This association was a constant finding in this study. Exceptions to this occurred in cases where the tuberculosis was so minimal as not to involve vessels of sufficient caliber to be seen angiocardiographically.

Displacement of the heart and great vessels was commonly found in chronic pulmonary tuberculosis. In Figures 1 and 5, an upward displacement of the right and left pulmonary artery, respectively, is demonstrated. In chronic upper lobe disease this particular

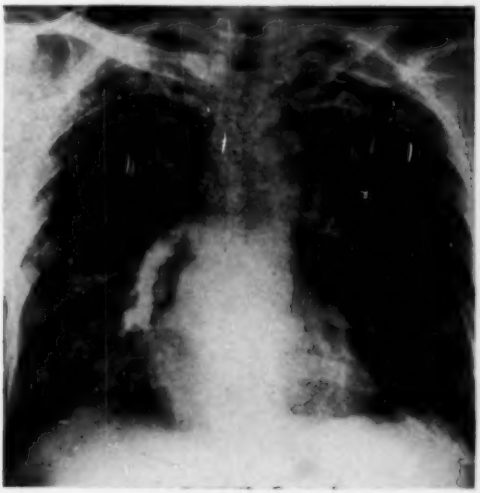

FIGURE 4 (F.J., No. 219): Angiocardiogram of a 24 year old Chinese man with caseo-cavernous tuberculosis of the right lower lobe. Marked avascularity of the involved lower lobe is evident.

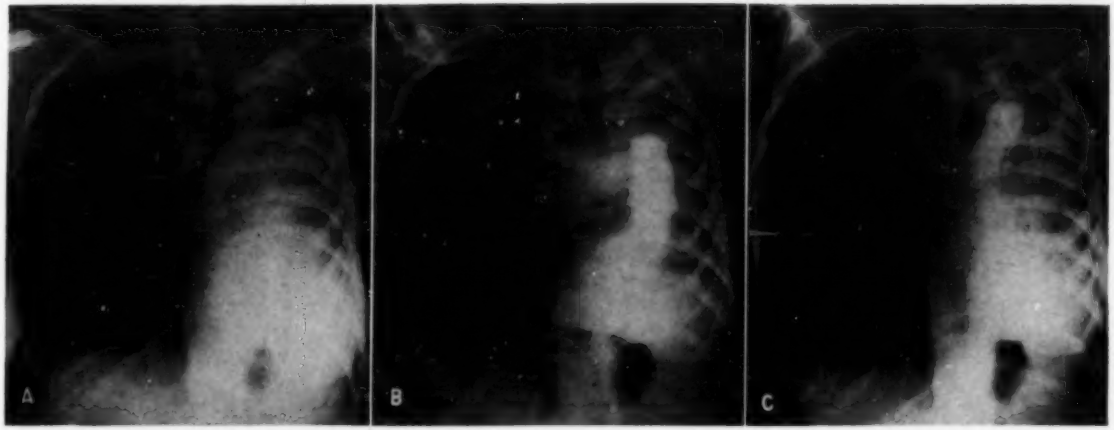

FIGURE 6 (J.I., No. 207): Left mediastinal tuberculoma causing recurrent nerve paralysis in a 28 year old Negro (operative and histological proof).—A: Conventional roentgenogram demonstrates a left mediastinal mass.—B: Angiocardiogram shows absence of opacification of left mediastinal mass (excluding the diagnosis of pulmonary artery aneurysm) and avascularity of the left upper lobe, probably due to obstruction by the tuberculoma.—C: Angiocardiogram shows opacification of a normal aorta ruling out an aortic aneurysm.

change was encountered frequently and was assumed to be secondary to fibrosis and retraction of the involved lobes. As a result of tuberculosis there may occur extensive unilateral pulmonary fibrosis with retraction of the heart, mediastinum, trachea and great vessels into the affected side a condition referred to as "fibrothorax." Thus a homogeneous shadow is formed which on a conventional roentgenogram cannot be differentiated into its components (Figure 6A). Angiocardiography delineates the cardiac chambers and great vessels (Figure 6B and 6C).[18] In this patient the heart had become rotated to the "right anterior oblique" projection. The right pulmonary artery was elongated and distorted while the left pulmonary artery (seen on end) was markedly reduced in caliber. Separation of the pulmonary vessels on the right suggest overdistension of the lung.

In silico-tuberculosis, pulmonary avascularity depends on the degree of parenchymal involvement of the combined disease. In the case studied, pronounced avascularity at the site of extensive silico-tuberculosis was seen. No detectable change in pulmonary vascularity was noted in one case of miliary tuberculosis with a complicating tuberculous pericardial effusion. Two other cases in this series also had tuberculous pericarditis with effusion. Films

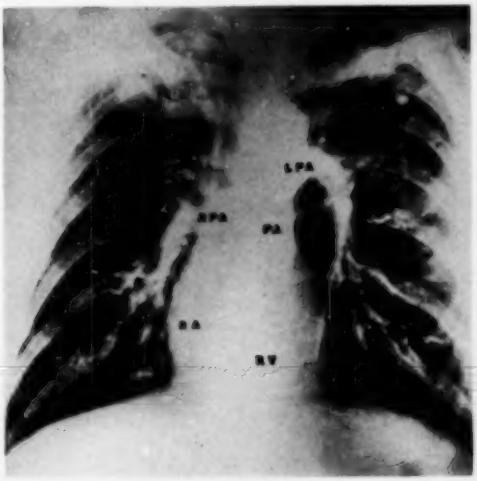

FIGURE 5 (H.J., No. 80): Angiocardiogram of a 48 year old man with fibrocalcific tuberculosis of both upper lobes. There is marked avascularity of the upper lobes with elevation of the left pulmonary artery.—RA: right atrium,—RV: right ventricle,—PA: pulmonary artery,—RPA: right pulmonary artery, —LPA: left pulmonary artery.

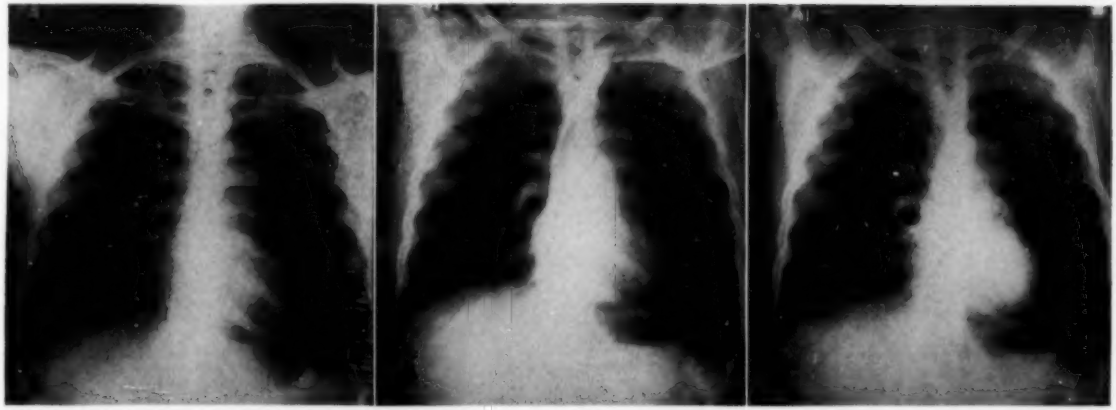

FIGURE 8 (M.S., No. 524 967): Far advanced fibro-cavernous tuberculosis of the left lung (fibrothorax) in a 42 year old woman treated with thoracoplasty.—A: Conventional roentgenogram showing retraction of the heart, trachea and mediastinum into the left chest forming an undifferentiated homogeneous density.—B: Angiocardiogram at 2½ seconds after the beginning of injection delineates the right heart chambers, the pulmonary artery and branches. The left pulmonary artery is not filled; the right pulmonary artery is elongated and enlarged. Its branches also appear enlarged, widely separated and extend into the opposite side of the chest, a pattern characteristic of overdistension of the right lung. The right heart and pulmonary vessels have been rotated to assume the position of the right anterior oblique view.—C: Angiocardiogram at 8 seconds after the beginning of injection shows opacification of the left ventricle, aorta and the brachiocephalic vessels.

of these patients were timed so as to opacify the heart chambers. In Figure 7 the border of the right atrium can be seen to be well within the cardiac silhouette. The intervening space represents the right atrial wall and the pericardial fluid surrounding the heart. In a repeat study at a later date this space was absent indicating resorbtion of the effusion.

Tuberculosis of the mediastinum or hilar area may sometimes simulate a malignant tumor or aneurysm on the conventional roentgenogram.[19] A 32 year old Negro with a recurrent nerve paralysis was found to have a left mediastinal mass which on conventional roentgenography could not be differentiated from an aneurysm or a malignant tumor (Figure 8A). There was no evidence of pulmonary tuberculosis. Angiocardiography revealed a diminution in the blood supply of the left upper lobe in association with the mass (Figure 8B). A film was taken at eight seconds after the injection of contrast substance so as to reveal the left cardiac chambers and aorta (Figure 8C). The mass did not opacify in either film thereby ruling out aneurysm. Subsequent operation and pathological study revealed this to be a mediastinal tuberculoma. Occlusion of the descending pulmonary artery of the right lung by tuberculous hilar lymph nodes was encountered in another case previously reported.[2] In the four other patients with tuberculous hilar and mediastinal lymphadenopathy, no vascular alterations were observed.

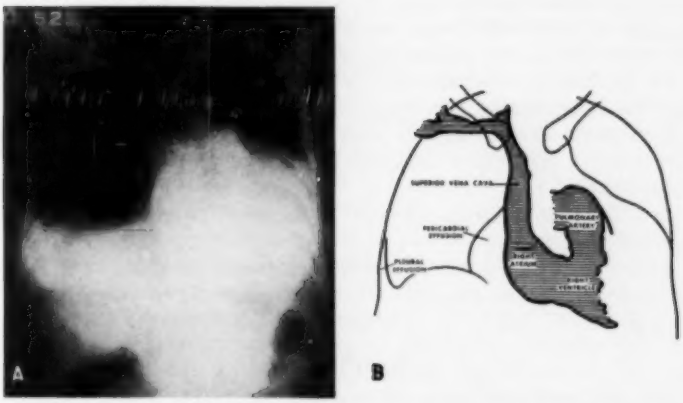

FIGURE 7 (M.S., No. 549 474): Fibro-cavernous tuberculosis of the right upper lobe in a 59 year old white woman.—A: Angiocardiogram shows opacification only at the origin of the ascending right pulmonary artery and marked avascularity of the upper lobe.—B: Tracing of A.

Discussion

Pathological studies of pulmonary blood vessels within an extending tuberculous process reveal early extensive thrombosis. The vessel walls become thick and fibrotic. Endarteritis obliterans may completely close the lumen reducing the vessels to thick solid cords in which subsequent organization and cicatrization may take place.[8-10] The avascularity associated with tuberculous lesions has been demonstrated roentgenographically by injection of radiopaque substances in both animal and human autopsy material.[11,12] Such post mortem visualization studies also demonstrate a lack of fine pulmonary arterial branching and definite diminution in size of vessels in areas of tuberculous tissue destruction.

The degree of avascularity was found to depend upon the amount of lung tissue involved. The more widespread the parenchymal tissue destruction, the more marked was the avascularity. The influence of these changes on the efficiency of respiration was shown to result in diminished pulmonary function by Cournand and Richards.[20] No changes in vascularity were noted in diseased areas where the involved vessels were too small to be seen angiocardiographically. This was true where lesions were small and discreet as in the case of miliary tuberculosis. Similarly, Birkelo and Brosius failed to demonstrate vascular changes in miliary tuberculosis by post mortem roentgenographic injection studies.[13]

Angiocardiography served to delineate the heart chambers and great vessels in fibrothorax, usually showing rotation, displacement and distortion of these structures (Figure 6B and 6C). The displacement of pulmonary arterial trunks secondary to fibrosis and retraction of the disease area has also been visualized (Figures 1 and 5). Cardiac chamber visualization has been utilized to establish the diagnosis of pericardial effusion (Figure 7).[21] This has proved to be of considerable practical value in patients in whom the clinical, laboratory and roentgenographic findings are not diagnostic.

As illustrated in Figures 8B and 8C, angiocardiography may also demonstrate alterations in pulmonary vascularity caused by enlargement and invasion of tuberculous mediastinal and hilar lymph nodes. Masses in these areas may simulate aneurysm. Angiocardiography is most helpful in ruling out the latter.[22,23]

SUMMARY

The angiocardiographic findings in 91 tuberculous patients studied during life are presented. Diminished vascularity of varying degrees was found to occur in local areas of the lung involved by tuberculosis. The degree of avascularity depended upon the amount of lung tissue involved. The more widespread the paren-

chymal lesion, the more marked was the avascularity. Displacement of vessels secondary to fibrosis and retraction was encountered frequently. In tuberculous fibrothorax, angiocardiography delineates the cardiac chambers and great vessels, often revealing marked distortion and displacement of these structures. By contrast visualization, hilar and mediastinal masses may be differentiated from aneurysm. In two instances tuberculous hilar and mediastinal adenopathy resulted in obstruction of major branches of the pulmonary artery. Finally, the use of angiocardiography in the detection of pericardial effusion is illustrated.

RESUMEN

Se presentan los hallazgos angiocardiográficos en 91 tuberculosos estudiados durante la vida. Se encontró disminución de la vascularización en grados diversos en las áreas invadidas por la tuberculosis. El grado de vascularización depende de la cantidad de pulmón comprometida. Mientras más extensa la lesión parenquimatosa más marcada es la vascularización.

A menudo se encontró desplazamiento de los vasos en los casos de fibrosis y retracción. En el fibrotórax tuberculoso la angiocardiografía delinea las cámaras cardiacas y grandes vasos frecuentemente revelando marcada distorsión y desplazamiento de estas estructuras. Por contraste, por la visualización de hilio y mediastino se pueden diferenciar del aneurisma. En dos casos la adenopatía hiliar y mediastinal produjeron obstrucción de ramas mayores de la arteria pulmonar. Finalmente se ilustra el uso de la angiocardiografía para descubrir la presencia de derrame pericárdico.

RESUME

Les auteurs présentent les angiopneumographies qui ont été faites chez 91 malades tuberculeux durant leur vie pendant une période de 14 ans. Ils ont constaté que les régions du poumon atteintes de tuberculose étaient le siège d'une diminution de la vascularisation, de degré variable. Cette diminution de la vascularisation est en rapport direct avec l'étendue du tissu pulmonaire atteint. Plus il y avait d'ensemencements tuberculeux, plus la vascularisation était déficiente. Il y à également fréquemment chez ces malades un déplacement secondaire des vaisseaux, dû à la fibrose et la rétraction. Dans le fibrothorax tuberculeux, l'angiopneumographie permet de délimiter les contours du coeur et des gros vaisseaux, et révèle fréquemment un déplacement et une déformation très marquée de leur profil. Des images anormales des hiles et des masses médiastinales peuvent être différenciées de l'anévrysme. Dans deux cas, les hiles atteints de tuberculose et des adénopathies médiastinales étaient dûs à l'obstruction des branches

principales de l'artère pulmonaire. Enfin, les auteurs mettent en évidence l'intérêt de l'angiopneumographie pour la découverte d'épanchements péricardiques.

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A Case of Tuberculous Pericarditis Associated With a Primary Pulmonary Tuberculous Infection*

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This report concerns a case of tuberculous pericarditis in a patient having a recent primary tuberculous infection in the lung and also the lymph nodes around the hilum.

The case is that of L. S., a 19.1-year-old Negro school girl, born in Chicago in 1922. There were no childhood illnesses of consequence, but her story was incoherent and unreliable so far as early history was concerned.

In December 1940, however, she was sent to the Cook County Hospital with a diagnosis of scarlet fever. She was there one week and was sent home—obviously as a result of a change in diagnosis. In a few days she developed a pain in the left side of her chest and went to the Municipal Tuberculosis Sanitarium (Wacker Drive Clinic) where tuberculosis was diagnosed and fluid was removed twice within one week. She was sent back to the Cook County Hospital on April 1, 1941, but was transferred to the Municipal Tuberculosis Sanitarium on April 12, 1941. At that time she complained of a slight cough, slight dyspnea, and a slight loss of weight. She was not acutely ill. On physical examination, there was little of significance outside of the chest and heart.

The chest was flat, excursion was poor, fremitus normal. Dullness was present in the lower half of both lung fields. The breath sounds were weak, and scattering subcrepitant rales were present in the whole lower half of the chest.

On the roentgenogram there was a tremendous enlargement of the heart area extending out nearly to the pleura on both sides.

The diagnosis was not certain, but lay between a post-scarlatinal or tuberculous pericarditis with associated pulmonary tuberculosis and pleural effusion. The electrocardiogram was reported to be compatible with that of pericarditis.

There was a regular rate of 130 per minute, a regular rhythm, low voltage, and inverted T-waves in all leads.

The Montoux test was strongly positive, but all sputum tests were negative. There were a few granular casts and leukocytes in the urine, and 100 mgs. albumin. The red cell sedimentation by Cutler's method was 13 mm. in 30 minutes. There was significant secondary anemia (3.7 rbc. and 55 per cent Hb.), a mild leukocytosis (11.500), 75 per cent polys, 15 per cent stabs, a shift to the left, 8 per cent monocytes, marked anisocytosis, and "elliptical cell" anemia.

The pleural fluid was negative on all direct examinations (April 26,

*From the City of Chicago Municipal Tuberculosis Sanitarium Research Laboratories, and the State of Florida Tuberculosis Hospital System.

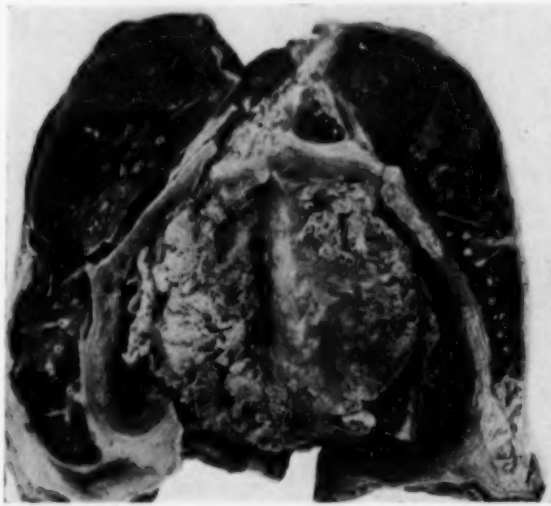

FIGURE 2

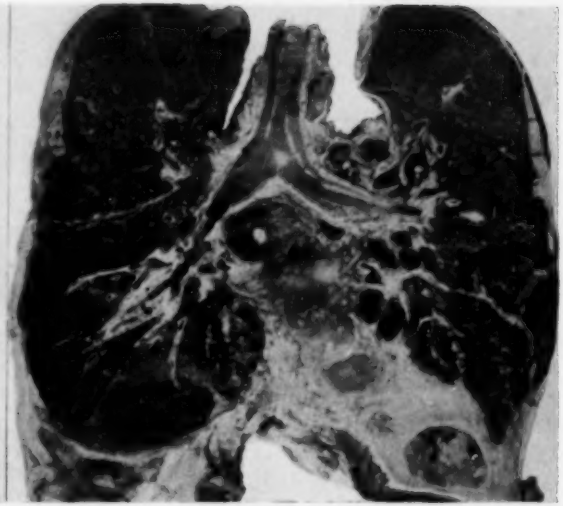

FIGURE 3

Figure 2: Anterior mediastinal sagittal section of chest showing lymph node tubercles in left anterior mediastinal nodes with thickened pleura, thickened pericardium, and shaggy heart.—*Figure 3:* Posterior mediastinal sagittal section with several lymph node tubercles in left superior and one in the inferior hilum node.

1941) but positive for tubercle bacilli on culture (May 22, 1941). The patient died on July 27, 1941, at 8:50 a. m., and the post-mortem was performed the same day at 1:00 p. m.

Necropsy

The body was that of a well-nourished, colored female, 19 years of age. The abdomen was distended one finger above the chest level.

The peritoneal cavity contained about 500 cc. of clear straw-colored fluid. The serosa was smooth throughout.

The diaphragms on both sides were at the fifth ribs. The left pleural cavity was completely obliterated by thick fibrous adhesions. The lower three-fourths of the right pleural cavity was similarly involved. The right apex was free of adhesions.

The pericardial sac contained about 30 cc. of clear straw-colored fluid. The heart was covered with a thick coat of organized and shaggy fibrin, in places 2 cm. thick. In a few areas the epicardium was exposed, and small 2-3 mm. yellow patches were visible. The pericardial membrane was thickened up to 2 cm. and was leathery in consistency. The lower lobes of both lungs were pushed laterally and posteriorly by the hugely distended pericardium.

The heart was not weighed, but did not appear to be enlarged. The myocardium was reddish-brown and firm. The endocardium of the valves was normal. The coronary arteries were normal, but were compressed by the growth in the pericardial sac.

The bacteriological examination of the pericardial fluid revealed the presence of tubercle bacilli.

In the anterior medial margin of the left upper lung lobe, there was a cluster of soft primary tubercles 3-8 mm. in diameter. In the lymph nodes toward the hilum and in direct relation to the lesions in the parenchyma, there were tubercles 3-4 mm. in diameter. In the greatly enlarged bronchopulmonary nodes, there were other caseous tubercles 3-5 mm. in diameter. In the superior tracheobronchial lymph nodes,

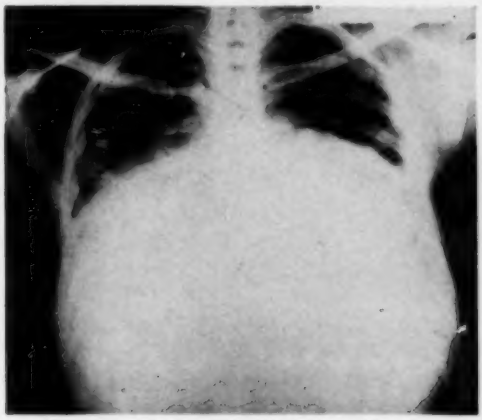

FIGURE 1: Roentgenogram of chest showing the characteristic shadow of pericarditis with effusion.

many tubercles 3-5 mm. in diameter, soft and recently encapsulated were also observed. A single tubercle in the hilum lymph node measured 7 mm. There was marked hyperplasia in the extensive lymph node involvement extending in and around the anterior part of the trachea where nodes came in contact with the pericardium. There was an occasional nodule 1-3 mm. in diameter throughout both lungs from apex to base, no doubt due to hematogenous dissemination.

The mucous surface of the trachea and main bronchi were normal.

The left pleura was thickened in several places by a fibrous pleuritis. The right was only slightly involved.

The gross appearance of all other organs were essentially normal except the cecum, in which there was an 8 mm. deep red, indurated ulcer.

The microscopic examination of most of the organs was not unusual. There was an ordinary miliary tuberculosis of the spleen and liver consisting of about two small epithelioid tubercles per square cm. A calcified 2 mm. nodule on the surface of the liver was characteristic of a calcified phlebolith. The lungs contained several foci in the anterior part of the left upper lobe with corresponding tubercles in the chain of lymph nodes towards and around the left side of the hilum, and one 7 mm. tubercle in the lymph node at the bifurcation. The process did not extend beyond this point except to produce foci of monocytes in some of the paratracheal lymph nodes and in the nodes at the angle of the innominate vein. Cross sections of the anterior (right) and posterior (left) coronary arteries did not reveal any damage to the main lymphatic trunks accompanying these vessels, although retrograde currents carrying a few bacilli may have taken place.

The character of the tubercles was much the same in the lungs and bronchopulmonary lymph nodes, but became less well encapsulated towards the hilum. The usual finding was a focus of caseation, early fibrosis

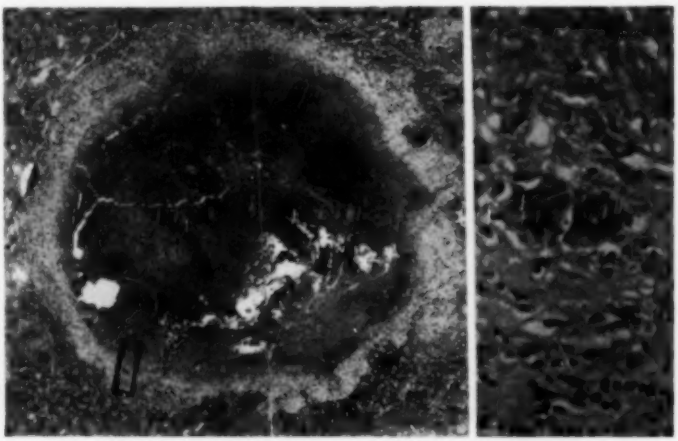

FIGURE 4 FIGURE 5

Figure 4: A characteristic lymph node tubercle with beginning capsule formation.—*Figure 5:* An enlarged field outlined by rectangle in Figure 4. Note the caseating, giant cells, and early fibrosis. This type of lesion is encountered in lesions of 1 to 2 years duration.

of the capsule with giant cells, and no calcification. The capsule of the oldest tubercles was characteristic of a 1½-2-year lesion.

The pericardium was covered with a layer of fibroblasts, monocytes, and many Langhans' giant cells. The central three-quarters was made of broad strands of hyalinized connective tissue. The epicardium was overlaid with fibrinous material, which was partly caseous, and was made up of cells and fibrocytes. An area at the apex of many square centimeters was caseous, extending 1-2 mm. into the myocardium. The myocardium was only involved superficially.

Discussion

The significant features of this case were the development and course of what appears to be an ordinary young adult type of primary tuberculous lesion with several blood stream episodes and the development of a marked fibrinous and fibrous pericarditis. While the date of the first infection is not obvious, we can be certain it was more than nine months before death, but probably not over two years. Since there were no tuberculin tests to show when allergy developed, the duration must be adjudged by the clinical history and the age characters of the lesions, which were adjudged to be 1½-2-years of age.

The next important clinical episode was the "pseudo-scarlet" fever, which may have been a primary tuberculous erythema or probably a manifestation of the first major shower of hemato-

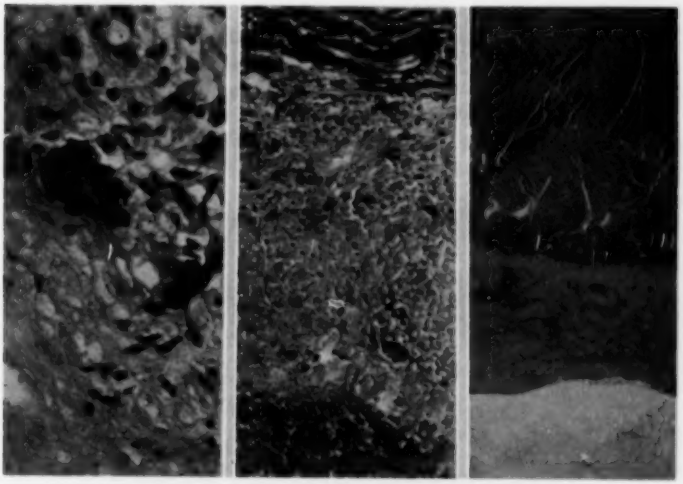

FIGURE 8 FIGURE 7 FIGURE 6

Figure 6: A low power microscopic photograph of heart and epicardium showing a tremendous thickening of the epicardium with caseous patches scattered throughout.—*Figure 7:* A more enlarged picture of the caseous and fibrotic regions.—*Figure 8:* A still more enlarged region revealing giant cells.

genous foci of tubercle bacilli. As there is no accurate description of these lesions, we must assume they resembled scarlatina.

The third and most important episode was the pericarditis. There are three routes by which tubercle bacilli may reach the pericardium, viz: First by the blood stream as part of the second stage of Ranke, as a generalized miliary, or as part of a polyserositis; second, by a direct extension through the pericardium from caseous tissues (lung, lymph nodes); third, by the lymphatics. Riesman,[1] Bialock and Levy,[2] Hemerson,[3] Beller, McMillan and Gonley,[4] and others emphasized these three general routes with certain variations.

Most authors agree that the hematogenous origin is usually as a part of a generalized hematogenous systemic disease with bacilli reaching the pericardium by way of the blood stream. The direct extension is usually an abrupt rupture into the pericardium with a *sudden* development of symptoms. The retrograde lymph flow is more likely to produce a gradual development of symptoms as in the case reported. Heimann and Binder's work[5] in Banto Negroes practically proves the point because they state that while eight of their 30 cases developed generalization before death, in none was the pericarditis a part of a generalized miliary process. Nor was there any having a direct extension into the pericardium. Retrograde lymph flow, therefore, seems to have accounted for most of pericardial localizations. That is the probable route of the infection in the case described.

In this case, there seemed to be possibilities of a retrograde extension from the left broncho-pulmonary nodes to the left superior tracheobronchial into the mediastinal nodes below the innominate vein and thence back through the right lymphatic trunk until a spill into the pericardium resulted.

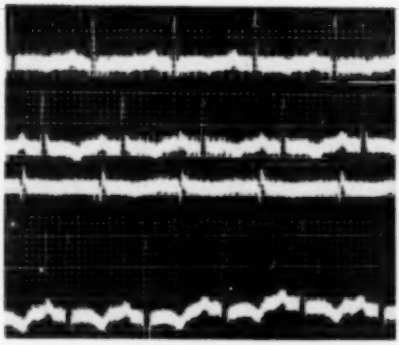

FIGURE 9: An electrocardiogram with somewhat low voltage and inverted T waves in all leads.

The character of the fibrosis is similar to that of a pleuritis occasionally seen in primary infections and seems to occur during a primary infection before allergy has developed.

Of the many other cases of tuberculous pericarditis in our material, this is the only one having the thick "leathery" fibrous tissue. Most of the cases had evidence of a direct extension of caseation from the lymph nodes of the pleura with a purulent type of pericarditis. Others had thin, fibrous adhesions. In one case, the disease was part of a generalized miliary tuberculosis originating in the kidney.

Shore[6] has worked out the lymph supply of the heart in sheep, rabbits, and Bantu Negroes. According to him, there are two lymphatic trunks, a right or anterior trunk that arises along the branches of the right coronary, and runs beneath the serosa on the anterior aspect of the aorta, to the anterior mediastinal nodes. (One of these nodes is in the region of the ductus Botalli.)

The left or posterior trunk follows the left coronary, passes under and posterior to the right pulmonary artery and between the pulmonary artery, aorta, and superior vena cava, to drain into the right inferior tracheobronchial nodes. These nodes have been called the caval nodes by Shore.

A tuberculous lesion in any of these nodes might be the source of an infection as a result of retrograde lymph flow. Lymph drainage is prone to become retrograde when the channels become blocked.

SUMMARY

A case of heavy fibrous tuberculous pericarditis has been described, wherein the origin of the pericarditis was thought to be due to a retrograde lymph flow as a part of a primary tuberculous infection in the lungs and lymph nodes.

RESUMEN

Se ha descrito un caso de pericarditis tuberculosa fibrosa, en el que se creyó que el origen de la pericarditis se debió al flujo linfático retrógrado, parte de una infección tuberculosa primaria en los pulmones y ganglios linfáticos.

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Metastatic Carcinoma of the Pericardium*

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Disease of the pericardium, regardless of etiology is relatively uncommon. White[1] states that it occurs in approximately 5 per cent postmortem material and that in one-half to two-thirds of these cases, it is encountered as an acute condition. Smith and Willius[2] reported an incidence of 9.2 per cent among 15,363 necropsied cases. Malignant disease of the pericardium, primary or secondary is uncommon. Secondary tumors are more frequent; carcinoma predominating. Most of the metastases are blood borne with the heart and pericardium part of a generalized dissemination. Less often discrete solitary metastases or direct invasion from adjacent viscera occur. The primary growth may occur in any organ. The secondary pericardial lesions, like those found any other place are usually small, discrete, firm and whitish nodules. Diffuse infiltration of the pericardium may occur. Fibrinous pericarditis may result from metastatic lesions. Occasionally fluid collections occur, often sanguinous. Accumulation of hemorrhagic pericardial fluid is always suggestive of a pericardial or subpericardial malignant process. Pericardial adhesions or complete pericardial symphysis with or without constriction may occur.

It may be seen that malignant disease of the pericardium produces the same type of response in the pericardium that may be found with other diseases. Most neoplastic involvement is asymptomatic, the lesions being found as incidental metastases at necropsy. Occasionally the involvement may produce effects which suggest pericardial disease. Where this occurs in the presence of a known primary growth in the body, suspicion of malignant disease of the pericardium should arise. Yater[3] in an excellent review of tumors of the heart and pericardium divided the cases into two groups. Those in which the clinical types were not suggestive of tumor of the heart. In this group were included the asymptomatic cases, those with symptoms of terminal cardiac embarrassment, those with symptoms of congestive heart failure, subacute bacterial endocarditis, and sudden death. In the second clinical group the clinical picture suggested tumor of the heart

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with the occurrence of symptoms or signs of cardiac involvement in patients with known or suspected malignant disease. Here the manifestations were due to pericardial effusion (often hemorrhagic), x-ray evidence of a localized enlargement of the heart, or an irregularity of cardiac contour, cardiac arrhythmia including auricular fibrillation and heart block, and myocardial insufficiency. While malignancy is a rare cause of pericardial disease, the pericardium itself is not uncommonly involved in neoplastic cases. Scott and Garvin[4] in an excellent review of tumors of the heart and pericardium, reported an incidence of 10.9 per cent among 1,082 patients with malignant disease. Of their 118 cases with cardiac involvement, there were 61 with secondary malignancy of the pericardium, in 22 per cent of which the pathological process was acute. A simple effusion was found in seven, bloody fluid in seven, purulent pericarditis in three, and 11 cases exhibited a serofibrinous reaction. Myocardial insufficiency was found in seven of their cases, but none showed the "constriction" syndrome. Sarcoma and lymphoblastoma involved the pericardium relatively more commonly than carcinoma, of which there were more cases. Smith and Willius[2] reported no case of effusion due to malignancy in their series. Yater[3] noted small pericardial effusions in his series of 11 cases. Constrictive pericarditis due to malignancy of the pericardium is very rare. Smith and Willius[2] found an incidence of 2.8 per cent malignancy in 144 cases of chronic adherent pericarditis, encountered among 373 cases of pericardial disease. Of their four cases none showed the clinical picture of concretio. Scott and Garvin[4] found one case of obliteration of the pericardial cavity by tumor and two by fibrous tissue, but none show the picture of compression. Leaman's[5] case of constrictive pericarditis due to metastatic bronchogenic carcinoma showed the well developed syndrome. Wallace and Logue[6] reported an interesting case of bronchogenic carcinoma producing constrictive pericarditis and developing a clinical picture resembling right heart failure. The ECG showed typical pericarditis pattern. At necropsy the heart was found to be completely encased in tumor tissue, the infiltrated pericardium being over 2.5 cm. in thickness. This case is remarkably similar to Case II.

Any primary malignant tumor may spread to the heart, however, most cases of metastatic invasion of the heart and pericardium are associated with either primary or secondary intrathoracic tumor. Lymburner[7] found an 88 per cent incidence of intrathoracic neoplasm in 52 cases of secondary malignancy of the heart encountered in 8,500 postmortem examinations at the Mayo Clinic. In 72 per cent of these, the lung was involved. Scott and Garvin[4] found cancer of the breast and bronchus to be the most common

primary sites metastasizing to heart and pericardium. In their series each was responsible for 35 per cent of the cases. The incidence of pericardial involvement in bronchogenic carcinomas has been found to vary from 2 to 26 per cent. The incidence[8] of pericardial involvement in cancer of the breast[9] varied from 4.52 to 18 per cent and in pancreatic malignancy[10] from 3 to 6 per cent.

In a total of 6,257 postmortem examinations performed at the Department of Pathology of the Queens General Hospital in 12 years, 1,032 cases of epithelial tumors were found. These only represented 17 per cent of all tumors found. The incidence of the primary sites of malignancy is shown in Table I.

A total of 31 cases of carcinoma were metastatic to the pericardium. The various types of pericardial lesions encountered are shown in Table II.

TABLE I

Primary Carcinoma	No. of Cases	Pericardium Involvement Per cent
Lung	177	13
Breast	34	6
Pancreas	67	3
Esophagus	35	3
Uterine-Cervix	5	6

TABLE II

Type of Lesion	Number of Cases
Simple Effusion	3
Bloody Effusion	5
Cloudy Effusion (purulent?)	1
Obliterative Pericarditis	6
Constrictive Pericarditis	1
Nodules	3
Direct Extension	12

Case Reports

Case I: C.K., age 42, sex female, was admitted to Mary Immaculate Hospital August 26, 1943 and expired September 10, 1943. The chief complaint was pain in the back and shortness of breath, of three months duration. There had been a moderate weight loss. On admission there was considerable respiratory distress. The respirations were 36, the pulse thready at 120. There was cyanosis of lips and nail beds and the neck veins were distended; blood pressure 84/74. The pulse was paradoxical. The liver was

palpable four fingers below the costal margin and was tender. There was the suggestion of ascites. Third heart sound audible. There was dullness at the left base with absent breath sounds, P-2 was louder than A-2. An x-ray film of the heart showed a generalized enlargement with a pear shaped configuration. The vessels in both hilar areas were considerably enlarged (see Figure 1). The ECG showed a low voltage T-1 and QRS. T-2 and T-3 were inverted. Chest leads were normal. The venous pressure was 20 cm. of water. Clinical diagnosis: Pericardial effusion, etiology undetermined. The course was progressively downhill with increasing cyanosis, orthopnea and distention of the neck veins. Fever remained elevated 101 to 104 degrees F. The blood count showed 92 per cent hemoglobin and 11,600 WBC; 85 per cent polymorphonuclears. The urine showed a 1+ albumen. Blood culture was negative. The urea nitrogen 18.9 mgm.

At necropsy the patient was fairly well-developed and well-nourished. On opening the thorax the under-surface of the sternum was studded with whitish, hard nodules, from pinpoint size to 2½ cm. in diameter. In the area of the heart a large bulging purplish red sac protruded forward and spread laterally, pushing back the lung surfaces. The pericardium and the parietal and visceral pleura too were studded with the above described white nodules. On opening the pericardium, a dark reddish fluid flowed freely and measured about 750 cc. The surface of the heart and the great vessels were studded with nodules. There were no adhesions present. Microscopic study revealed the primary growth in the pancreas. The final diagnosis was: Primary carcinoma of the pancreas with generalized metastases, a metastatic hemopericardium.

Case II: H.W., a 39 year old white housewife was first admitted to Queens General Hospital October 3, 1944. The chief complaint was a temperature of 101 to 102 degrees F. for a month, shortness of breath of a month's duration; soreness and swelling in the left cervical region for two to three days. She appeared feverish but not acutely ill. Pulse: rapid and regular, temperature 102.76 degrees F. P-2 louder than A-2. Tenderness in the left cervical region with prominent veins. There was a scraping pericardial sound during systole which was interpreted as a pericardial rub. The diagnosis was acute cervical lymphadenitis and acute pericarditis. The WBC was 14,000 with 78 per cent polymorphonuclears. Venous pressure and circulation times were normal. Following a course of sulfadiazine, the pericardial rub disappeared, the ECG on third and thirteenth days of hospitalization showed low voltage QRS in all the leads, inverted T1, T-2 and T-cf 1, 2 and 4. The x-ray film showed a normal contour to the heart with slight increase in size to the left. There was a minimal infiltrate in the periphery of the right upper lobe which was interpreted as arrested pulmonary tuberculosis (see Figure 2). Kymographic study showed normal cardiac pulsations. The temperature subsided and the patient was discharged.

Her second admission was on February 7, 1945 and she expired April 20, 1945. Since the last admission she had noted progressive exertional dyspnea, orthopnea and a heavy feeling in the chest which did not radiate. She had a cough of some 10 days duration with blood streaked sputum. On examination the patient was well-nourished, temperature 102 degrees F., cyanotic, orthopneic and 2+ pretibial edema. The neck veins were full; no adenopathy was found. The pulse was small and rapid, the rate 120. There was increased precordial flatness to percussion.

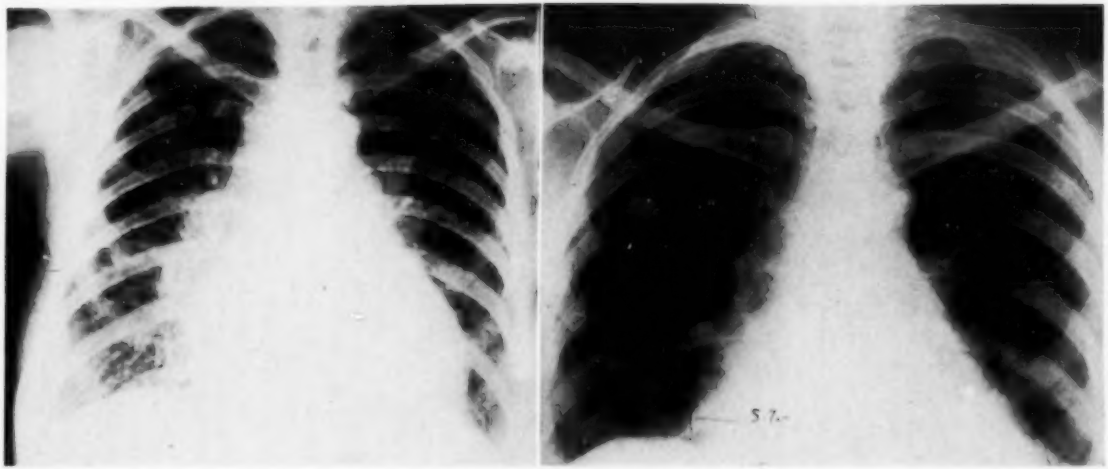

FIGURE 1 FIGURE 2

Figure 1: Chest x-ray shows heart enlarged and pear-shaped, pulmonary congestion due to the enlarged pulmonary veins and their branches.—*Figure 2:* Heart is enlarged to the left, the contour is normal. Note the solitary infiltrate over R-2.

The heart sounds were distant, P-2 louder than A-2, no murmurs and there was an apical 3-rd sound interpreted as a pre-systolic gallop. The tender liver was now palpable three fingers below the costal margin. Blood pressure 105/80, the ECG was the same as on the first admission, and was interpreted as compatible with pericarditis, this remained unchanged, except for a further drop in voltage of the QRS complex and the T waves. The venous pressure was now 16 cm. of water; the arm to tongue circulation time 40 seconds, arm to lung 19 seconds. The chest x-ray film showed a slight increase in size of the right upper lobe lesion. A barium study of the esophagus was normal. Kymography was interpreted as showing feeble pulsations. A normal variation in the contour of the heart was noted on inspiration and expiration films. The blood counts were normal. The sedimentation rate varied from 12 to 20 mm. per hour. The blood chemistry was normal. Fever was continuous, never exceeding 102 degrees F. While in the hospital she developed right pleural effusion which was tapped on five different occasions. The fluid showed a specific gravity of 1,012 to 1,014, fibrin, polymorphonuclear cells and lymphocytes, the former predominating. The fluid was always sterile and repeated search for malignant cells was negative. She did not react to 1 milligram of old tuberculin. Pericardicentesis produced no fluid. Treatment consisted of oxygen, diuretics, digitalis and low sodium diet. There was a slight increase in size in the right upper lobe lesion, and it was felt that this was a tuberculous lesion which had become reactivated, however, 22 gastric concentrates and cultures were repeatedly negative for tubercle bacilli. Because of the latter finding and the negative tuberculin test, tuberculosis as an etiological agent for this picture was then considered more remote. For this reason it was assumed that perhaps the lesion in the right upper lobe might represent primary carcinoma with metastasis to the pericardium. The patient expired in pulmonary edema.

Necropsy surgeon, Dr. R. Altman: The body is that of a 50 year old well-developed, well-nourished white female. There are 500 cc. of somewhat cloudy fluid in the right pleural cavity. The left pleural cavity is partly obliterated by fresh adhesions. The heart is shifted to the left. The pericardium is considerably thickened and nodular. The pericardium is firmly adherent obliterating the pericardial cavity. No fluid is present. The liver edge extends 3 cm. below the costal margin on the right. On section of the right lung, a poorly circumscribed firm gray mass is seen at the periphery of the upper lobe. This mass measures about 3.5 cm. in diameter, is moderately firm and shows minute areas of degeneration. The mucosa of the uppermost second-order branch of the right upper bronchus stops abruptly about 5 cm. from the periphery, to be replaced by tumor tissue, representing the primary tumor. Several other nodules of tumor tissue, measuring up to 1 cm. in diameter, are seen at the base of the right lower lobe which is firmly adherent to the diaphragm. The latter is studded with tumor nodules. Several hemorrhagic infarcts are seen in all of the lobes and antemortem emboli are plugging the larger vessels on each side. The pericardium is markedly thickened and studded with grayish-white transluscent nodules measuring up to 0.3 cm. in diameter. Complete pericardial symphysis is present. The lumen of the superior vena cava appears to be narrowed due to encroachment of tumor tissue upon the adventitia. The myocardium is normal.

Diagnosis: Bronchogenic adenocarcinoma, right lung with metastasis to pericardium—adhesive and constrictive pericarditis.

Comment

Neoplastic pericardial disease is most often asymptomatic. Pain is rare, but when present, is usually due to pleural rather than to pericardial involvement. Likewise dyspnea even when the pericardium is sufficiently involved to produce it, can often be ascribed to pleural, pulmonary or associated mediastinal involvement.

The clinical syndromes of pericardial effusion with some degree of cardiac tamponade, and of constrictive pericarditis lend themselves readily to recognition. However, because of the relative rarity of malignancy as a cause for these, some other etiology is usually given pre-eminent consideration. Purulent pericarditis has been reported complicating malignancy in the body as a result of perforating carcinoma of the esophagus. Even pyopneumopericardium has been reported with the latter. Pericardial symphysis is much more common than constrictive pericarditis, and is in the most part an incidental complication discovered at postmortem examination. We have encountered four cases of obliteration of the pericardial cavity and one of typical constrictive pericarditis. An interesting case of anterior pericardial symphysis with loculated posterior pericardial effusion due to metastatic carcinoma is mentioned by Yater.[3] We have encountered one case of bronchogenic carcinoma where the entire anterior pericardium was adherent and the posterior portion of the cavity free. These are mentioned merely to illustrate the diagnostic dilemmas they may entail. ECG changes illustrative of pericarditis are minimal in malignancy of the pericardium unless there is sufficient degree and types of involvement. The occurrence of any disturbance of rhythm in a known case of malignancy should always throw suspicion on the probability of heart involvement. Mohaim[11] has reported several instances of paroxysmal auricular fibrillation and flutter in patients with metastatic neoplasm of the pericardium. They attribute the arrthythmas to irritation of the epicardial nerve bundles. One of our cases with sanguineous pericardial effusion due to metastatic bronchogenic carcinoma showed auricular flutter. No myocardial involvement was noted on routine examination, but it is possible that some other factor may have been responsible for the arrhythmia.

SUMMARY

1) Malignancy of the pericardium is uncommon. It is usually asymptomatic, and part of a generalized dissemination. Associated symptoms are usually ascribed to the primary malignancy itself.

2) A case of constrictive pericarditis due to metastatic bronchogenic carcinoma has been presented in detail.

3) A case of sanguineous pericardial effusion due to malignancy has been noted.

4) The necropsy incidence of pericardial involvement by tumor has been listed and the lesions discussed.

5) Several recognizable syndromes can occur.

6) In the presence of known suspected malignancy, the picture of right heart failure should always stimulate search for evidence of pericardial effusion or constriction. These need not be confused with the striking clinical picture seen with lymphangitic cancer of the lungs and subacute cor pulmonale. The occurrence of arrhythmias should suggest epicardial involvement. Because of the frequency of bronchogenic and breast carcinoma and their high incidence of pericardial involvement, special attention should always be given in these cases to the pericardium. Hemorrhagic pericardial effusion should be considered neoplastic until proved otherwise.

RESUMEN

1) Las neoplasias malignas del pericardio no son comunes. Generalmente son asintomáticas y son parte de una diseminación generalizada. Los síntomas asociados son generalmente atribuídos al tumor primario mismo.

2) Se presenta en detalle un caso de pericarditis constrictiva debida a carcinoma bronquiogénico metastásico.

3) Se anota un caso de derrame pericárdico sanguinolento debido a neoplasia maligna.

4) Se apunta la incidencia del compromiso pericardiado por el tumor y se discuten las lesiones.

5) Pueden ocurrir algunos síndromes reconocibles.

6) En presencia de sospecha de malignidad el cuadro de insuficiencia derecha debe conducir a la búsqueda de derrame pericárdico o constricción. Estos necesitan no ser confundidos con el sorprendente cuadro clínico que se ve en el cáncer linfagítico del pulmón y en corazón pulmonar subagudo. La presencia de arritmias debe sugerir invasión del epicardio.

A causa de la frecuencia del cáncer bronquiogénico y del carcinoma de la mama y su alta incidencia de invasión del pericardio, se debe prestar especial atención en estos casos al pericardio. El derrame pericárdico hemorrágico debe considerarse como neoplásico mientras no se demuestre lo contrario.

RESUME

1) Le cancer du péricarde n'est pas fréquent. Il est généralement silencieux et ne réalise qu'un élément de dissémination.

2) Les auteurs rapportent en détail un cas de péricardite constrictive causée par la métastase d'un cancer bronchique.

3) Ils ont noté un cas d'épanchement péricardique hémoragique d'origine cancéreuse.

4) Ils ont consigné à l'autopsie les conséquences de l'atteinte du péricarde par la tumeur et ont discuté les lésions.

5) Ils ont pu avoir affaire à différents syndromes individualisés.

6) Lorsqu'on sait qu'il existe un cancer, le tableau clinique d'une insuffisance ventriculaire droite doit tojours engager à rechercher l'existence d'un épanchement péricardique ou d'une péricardite constrictive. Ces constatations ne doivent pas être confondues avec le tableau clinique tout à fait frappant du cancer lymphangitique du poumon associé à un coeur pulmonaire aigu. L'apparition d'arythmies doit faire penser à l'atteinte péricardique. Etant donné la fréquence des cancers broncho-pulmonaires, et la fréquence de l'atteinte péricardique, il faut toujours dans ces cas examiner avec soin le péricarde. Un épanchement hémorragique du péricarde doit être considéré comme cancéreux tant qu'aucune autre étiologie n'a pas été prouvée.

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Tuberculosis of the Genito-Urinary Tract

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One of the main considerations in the problem of genito-urinary tuberculosis is the constitutional aspect of the disease. The urologist is aware of the fact that when the genito-urinary tract is involved, tuberculosis has been pathology producing some place else, either previously or at the same time. He also knows that the treatment of this disease calls for serious consideration of this fact and it cannot be too strongly insisted upon that possible surgery, directed at the elimination of any tuberculous lesion involving the urinary tract, be followed by a period of care equal to sanatorium conditions.

There is another general consideration of genito-urinary tuberculosis which may easily be overlooked, namely that of the infectiousness of the urine or the discharges from draining sinuses. I mention this before engaging in a general discussion in order to give this some emphasis.

Genito-urinary tuberculosis may appear at any age and in either sex, but in general it occurs mostly within the period between 20 and 40 years. Members of both sexes are frequently victims. The incidence in various sanatoria and municipal tuberculosis institutions varies from 1.5 per cent to 50 per cent, depending upon the type of case the particular institution admits. The method of involvement is usually considered hematogenous and whether the urinary tract or the genital tract is involved first is a toss-up. It is usually a unilateral infection, but unfortunately is bilateral in too large a percentage of cases.

Types of Tubercle Bacilli: The types may be identified by observing the effect on guinea pigs, rabbits and fowl and also by the appearance of cultures. Petroff believed that all acid-fast bacilli are the same family and that the particular type we happen to find is dependent mainly upon the evolutionary influence of the environment by which the organism has been influenced. Insofar as recognizing the presence or absence of these bacilli is concerned, the method used depends upon the nature of the facilities available where the patient happens to be. The acid-fast stain of sedimented urine is only as dependable as the ability of the technician doing the stain. Guinea pig studies, broadly, are probably the most reliable in most communities. Cultural studies are enthusiastically advocated by many workers.

Pathology: Pathology should be divided into the acute and chronic forms. The acute form is a miliary involvement of whatever organ happens to be attacked as a result of a general acid-fast septicemia. This is a terminal condition and, before streptomycin, was of no interest from the standpoint of management. The chronic forms, however, are the ones which we are called upon to treat and should be divided into the necrotic type, the proliferative, and granulomatous type, and insofar as the kidneys are concerned, a so-called tuberculous nephritis. The necrotic type is a condition in which the tissue surrounding the tubercle formation becomes necrotic and the seat of calcium deposition or not, depending upon the individual. The proliferative type is one in which there is no appreciable necrosis, but the tissue involved becomes proliferative and results in the development of a much larger mass. Some kidneys develop to two or three times or more than their normal size as a result of this type of pathology. Tuberculous nephritis is an entity about which there is considerable controversy. Wildbolz believes this entity is one in which the kidney, as a result of tuberculous infection, is the seat of non-specific inflammatory changes in which there is an excretion of tubercle bacilli with no actual tubercle formation. Liberthal does not agree with this opinion.

Clinical Picture: There is no disease entity which has a more varied clinical picture than genito-urinary tuberculosis. Renal tuberculosis in most instances comes to the attention of the urologist because of uncontrollable and persisting frequency and dysuria. The next most frequent symptom is a persistence of renal infection. After this, the symptoms which bring the patient to the physician are divided pretty much equally between the symptom of pain over a kidney area with or without colic, albuminuria, hematuria, and tumor mass.

A typical picture, which is very simple, consists of frequency and dysuria associated with pain over one kidney area in a patient who has had tuberculosis in some other place in the body in whom we find acid-fast bacilluria. From this picture we have many variations, some with no symptomatology whatsoever to direct ones attention to the presence of renal tuberculosis, to those with extreme frequency and dysuria along with severe renal pain, the demonstration of acid-fast bacilli in the urine, and urographic demonstration of cortical destruction in the kidney involved. Cystoscopically, the bladder is nearly always the seat of a rather severe inflammatory, injected surface—usually hemorrhagic. The ureter on the side involved is usually definitely reddened and in many instances is gaping and described as golf hole in appearance. Ulceration of the bladder is not an infrequent

condition. The capacity of the bladder is usually markedly reduced and in some instances has been such a serious factor that perforation of the bladder has occurred by the beak of a cystoscope at the time of cystoscopy.

The urographic findings which we interpret to be indicative of tuberculous involvement are, first, a moth-eaten or irregular outline of a minor calyx in an early lesion and a generalization of this entity in the advanced lesion. The ureteral outline when irregular throughout its course is usually considered strongly suggestive of an acid-fast lesion. Calcification occurs in varying degrees in different individuals. Such instances occur as deposition of calcium in a ureter throughout its entire course, plus the kidney, so that when an x-ray film is taken the entire kidney and ureter are outlined by the calcium deposit in the tissues. This is a rare occurrence. In the patients who have had a low-grade and slow-developing process, we may see lobulated shadows, large in caliber, which represent a great amount of caseous material, filling a rather markedly distended kidney. Some of these cases eventually result in total occlusion of the ureter at some point in its course and a resultant so-called auto-nephrectomy. When this occurs we occasionally misinterpret the disappearance of acid-fast bacilli from the urine as indicative of improvement. The x-ray picture of the bladder itself is not pathognemonic. We can only recognize a small capacity bladder. Insofar as bacteriology is concerned we may have any type of mixed infection along with the acid-fast organisms, and these other organisms need to be given serious consideration and vigorous treatment. Tuberculosis of the prostate is suspected whenever, in an individual who happens to have renal tuberculosis, we feel an indurated, irregular consistencied prostate gland, with nodular outline. The vas is frequently thickened and in a so-called typical case it is beaded. Insofar as the scrotal contents are concerned, the epididymis is most frequently involved when a beaded or nodular type of epididymis is palpated one should think definitely of the possible presence of an acid-fast infection. The testicle is infrequently involved unless the individual has neglected himself for a long period of time.

Treatment: The treatment of renal tuberculosis resolves itself into general measures and surgery. We still are in controversey as to whether renal tuberculosis becomes arrested. One can only say that some individuals who have had a tuberculous kidney removed, have subsequently had the remaining kidney involved and after a period of sanatorium treatment they have engaged in gainful occupations. We see instances of acid-fast bacilluria in individuals who eventually become non-acid-fast under sana-

torium care. I once had the opportunity of observing three young women in the infectious disease section of Gallinger Hospital with acid-fast bacilli in their urine and clinical symptoms of renal tuberculosis, who all became free of acid-fast bacilli and returned to duty in a most vigorous general activity after a year of sanatorium routine. Having made these observations, it seems that there is justification for medically treating a so-called minimal lesion, namely one in which the symptoms are not progressive and one in which the roentgenological evidence consists of nothing more than slight localized involvement in one minor calyx.

In canvassing a number of authorities some years ago, I received various expressions of policy in regard to the indications for nephrectomy. The most vigorous was from Professor Hugh Young, who stated that the only misgiving he ever had insofar as removing kidneys with tubercle bacilli in the urine was that he had not removed some of them sooner than he did. Before the advent of streptomycin, it has been our policy not to do nephrectomy in an individual who had a minimal lesion which was not progressive. Any patient in whom progress was noted during a three-month period of observation was considered a candidate for nephrectomy. The problem of bilateral renal tuberculosis is one of common sense and judgment. The indication for nephrectomy in the presence of bilateral involvement is that of the extensive, acute symptom producing and rapidly advancing process in one side, along with a minimal, relatively symptomless lesion on the other, with the exception of the presence of tubercle bacilli or minimal involvement. If pulmonary or other terminal pathology exists, surgery is contra-indicated. Wherever other involvement is minimal, however, and the renal disease is most acute and extensive, nephrectomy is justifiable.

The advent of streptomycin has changed the whole problem and at present we are engaged in attempting to appraise its effect, so as to modify indications as to the type of treatment. We have seen spectacular and unbelievable changes in the urograms in patients with renal tuberculosis who have been treated with streptomycin or a combination of streptomycin and moogrol. Para-aminosalicylic acid has recently been added to our armamentarium. Our experience with some of these patients leads us to believe that at the present time we may definitely defer surgery unless we are unable to make the individual's urine free of acid-fast bacilli. We feel that even though we have eradicated all x-ray evidence of renal involvement, we are not justified in sending a patient back into general activity who has to carry a burden of hard work and who has not been rendered free of acid-fast bacilli in his urine. Where an individual may be able to accept his activi-

ties in such a way that he can have all the rest he needs, we feel he should be observed periodically.

Aureomycin has had some virtue in the control of bladder symptoms and in one case which we had an opportunity to observe, extensive tuberculosis in a solitary kidney has been favorably controlled by aureomycin after failure of four weeks intensive combination para-aminosalicylic acid and dihydrostreptomycin had failed. Streptomycin has been so spectacular that it has even been possible to perform uretero-sigmoidostomy successfully. The treatment of prostatic tuberculosis is also a controversial one. Some do prostatectomy, along with complete excision of the seminal tract, when there is involvement of this tract. When an epididymis is involved, we advise epididymectomy with or without removal of the testicle, depending upon the condition at the time of surgery. When extensive bilateral epididymo-orchitis exists, we have used pellate testosterone implantation in order to ease the acute endocrine imbalance which was resultant, with considerable satisfaction.

General Management: General management is probably the most important consideration in the handling of persons with tuberculosis of the genito-urinary tract. First of all, the individual himself must be convinced that his problem is a general one which will take time. Unfortunately there are no communities (or certainly few) which adequately take care of the individual who finds himself confronted with having had a kidney removed and yet has the responsibility of a family. This individual usually goes back to work and not infrequently comes down with subsequent involvement of his remaining kidney, and dies. This is a sad commentary on the present social policies in most large centers of population. Rest is the number one necessity. The individuals should not engage in physical activities other than those of moving around enough to get meals and go to the bathroom, plus being allowed in the sunshine and fresh air when the climate permits. Sunshine is valuable in the absence of pulmonary lesions, according to our observations. Statistically, the Southwest part of the United States is the most favorable area insofar as genito-urinary tuberculosis is concerned, and were it possible to send all genito-urinary patients there, the general result would probably be better than is the case where the patients are scattered throughout less favorable climatic surroundings. The peace of mind of the individual is another very important consideration. This must be adjusted and thought of as an individual problem. There is nothing so damaging as worry, generally,—and particularly insofar as this emotion and its relation to the tuberculous patient is concerned. Any device which takes up the attention of the patient is certainly

valuable. Insofar as food is concerned, a general nutritious diet is important, and with the exception of personal idiosyncrasies the diet should include milk in appreciable quantities, because of its calcium content, meat, eggs, and generally balanced nutritional regime. In addition to this, whole cod liver oil, in one form or another, has definite virtue and we advise it for all of our patients who can tolerate it. If under such ideal general management the lesion is progressive, we feel that even a minimal lesion should be surgically removed. Ultra-violet radiation of epididymitis and intra-cystic ultra-violet irradiation have been practiced with variable response.

The problem of uncontrollable frequency and dysuria is a chapter of its own and our plan for handling this most important situation is as follows:

(1) An attempt to eradicate all of the coincidental types of bacteria as satisfactorily as possible;
(2) Employment of anti-spasmodics of various kinds; and
(3) Administration of pyridium, caprokol, for the occasional beneficial effect on these symptoms.

Daily bladder instillations where the bladder will tolerate it of a nupercaine solution followed by one of the many other antiseptic or soothing solutions is followed out. Oelogomenol, cajandol, cod liver oil, solutions of phenol with 0.5 per cent and working up to 5 per cent, as suggested by Rovsing, are some of the instillations used. If, following all of these measures, the bladder is still uncontrollable and completely incapacitated, we have to consider cutaneous ureterostomy or nephrostomy, pre-sacral neurectomy, cordotomy, and prefrontal lobotomy. If streptomycin continues to be as wonderful as it seems at the present time, we may consider uretero-intestinal anastomosis as being definitely indicated in some cases.

Other Tuberculous Problems: Needless to say, appropriate handling of any other coincidental tuberculous lesion should be carried out. There is no condition that calls for team-work more than that of tuberculosis involving any part of the body. The social service aspect of tuberculosis definitely needs more in the way of resources than are now available.

SUMMARY

Tuberculosis involving any part or parts of the genito-urinary tract produces variable clinical pictures. We have at hand many measures which have resulted in a much more satisfactory theme of treatment than in former years. The future in chemotherapy holds out possibilities which have made all of us much more hopeful and happy than we have had an opportunity to be in the past.

RESUMEN

La tuberculosis de una parte o partes del aparato génitourinario produce cuadros clínicos variables. Contamos ahora con muchas medidas que han resultado en un tratamiento mucho más satisfactorio que en años anteriores. El futuro de la quimioterapia promete posibilidades que no existían en el pasado.

RESUME

La tuberculose en se localisant sur une ou plusieurs parties des voies génito-urinaires, crée des aspects cliniques divers. Nous avons actuellement à notre disposition des possibilités thérapeutiques nombreuses qui permettent des résultats bien supérieurs à ceux des années passées. La chimiothérapie nous réserve pour l'avenir des moyens d'action qui nous permettent d'être plus confiants que jamais.

Experiences with Dry BCG Vaccine in the Philippines*

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BCG vaccination as a prophylactic weapon against tuberculosis was initiated by the National Chest Center in February, 1948 with the donation of 1,100 vials of dry BCG by the Pasteur Institute of Paris.[1] Until the receipt of liquid vaccines from the New York Division of Laboratories through the United States Public Health Service in July, 1949 all vaccinations were done exclusively with the dry BCG. Before the program was started, however, tuberculin testing of school children and inmates of charity institutions had been done to gauge the extent of tuberculous infection among the city population, and to determine the most practical age groups in which to perform vaccinations.

It is the purpose of this paper to show our experiences with the dry BCG vaccine only in-so-far as methods and postvaccinal results are concerned, without evaluating its immunizing potency.

The Tuberculin Test

The tuberculin used was exclusively the PPD (Purified Protein Derivative) using the standard first dose of 0.00002 mg. in 0.1 cc. and standard second dose of 0.005 mg. in 0.1 cc. intradermally (Mantoux). The Vollmer patch tuberculin was used only experimentally in a group of children for comparative purposes, but was not favored as a routine measure because of the facility with which older children could remove it, the tendency of prickly heat formation especially during the hot months, and the discomfort borne by the child who remains without bathing for two or more days.

Early in 1949, 0.0002 mg. in 0.1 cc. was adopted as a single dose of tuberculin, or as a second dose following standard PPD I, as recommended by the Sub-Committee on Tuberculin Testing and BCG vaccination of the World Health Organization.[2] The titration experiments of Fourcolow, et al.[3] demonstrated the occurrence of false positive reactions with very large doses of tuberculin even in strictly non-contact groups. Standard PPD II being 250 times

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stronger than PPD I, this possibility was borne in mind, so that it was used only for further testing where the 0.0002 mg. PPD was negative in cases where residual or inactive tuberculosis was suspected. It was also used for postvaccination tuberculin testing, as allergy produced by vaccination has been found to be usually weaker than that resulting from a natural infection.[4] Interpretation of tuberculin reactions was made according to the standards set by the National Tuberculosis Association[5] whereby a reaction was considered positive only when there was erythema and induration of at least 5 mm. Erythema alone was discounted.

Newborns, and infants less than a month old who were confined in a nursery or otherwise known to be absolutely without tuberculous contact were vaccinated without a previous tuberculin test.

Plan of Investigation

To our knowledge, the Philippines is the first country in the Far East to use the dry BCG vaccine. Our main objective in its use was not that of an experiment, but to initiate what admittedly is an indispensable emergency measure in a highly infected population,[6] and to launch a campaign for the information of, and its acceptance by, the public.

The evaluation of dry BCG as an immunizing agent cannot be made within so short a time, and it is not improbable that whatever future results will be obtained may be open to question by those who would demand strictly controlled conditions wherein vaccinated and nonvaccinated groups being compared should be similar in sex, race, age, degree of exposure, intercurrent infections, nutrition, and other such variables of difficult control.

It is not possible, within the city of Manila, to find subjects with an entirely tuberculosis-free milieu. The Quezon Preventorium (White Cross), where we chose to initiate vaccinations, was perhaps the closest to a controlled group that we could find, if only from the fact that all inmates are x-rayed or fluoroscoped yearly, and all suspects removed from the institution. However, there is no real isolation from tuberculous contacts, because inmates are visited and allowed to be taken home on week-ends, and the parents or guardians of these children are usually tuberculous.

A relatively limited number of tuberculin-negative children outside of newborn infants, were eligible for BCG vaccination. Majority of those reporting in the BCG clinic were already tuberculin-positive. In the nurseries, very few mothers had had pre-natal chest x-ray films, so that investigation of household contacts could only be made when the child was brought for postvaccination tuberculin-testing. The parents, especially the mother, as

well as other members of the household, were urged by the vaccinating personnel to report for fluorography check-up at the National Chest Center.

Controls were taken at random from the list of babies born on the same day in the same hospitals where infants were being vaccinated, and who, because of refusal of the mother, or some other contraindication, such as fever, had not been vaccinated at the time. The mothers delivering in each of these hospitals usually came from similar social and financial brackets. No deliberate withholding of vaccination for purposes of acquiring a nonvaccinated control was done. With the widespread acceptance of BCG vaccination, it has been increasingly difficult to get a sufficient number of controls comparable with the number of vaccinated, unless one picked them from a random list of registered births.

Real isolation was not practised. It was emphasized to the parents that any contact with a tuberculous case should be avoided during the period before the child was tested with tuberculin and found positive. But such are the conditions in this country that this precaution was difficult of observance. The Quezon Preventorium, by arrangement with the president of the White Cross, was offered free to any parent who was willing to leave his child for the period when immunity from BCG was being developed. The vaccinated were usually asked to return after four weeks. Those found negative were retested the following month, and if still negative, were revaccinated.

All the reactors within the first 12 weeks were given certificates to show that they had reacted to tuberculin following BCG vaccination, and were asked to return six months after vaccination for another tuberculin test.

The Vaccine

One ampule of the dry vaccine contained 150 mg. of BCG in a glucose base[7] for cutaneous scarification. Each ampule carried its expiration date. All vaccines were refrigerated upon receipt.

Each new batch of vaccine was tested for viability, virulence and allergizing potency before actual use. Since our laboratory facilities were limited, and we were interested mainly in whether the concentration of vaccine being used for vaccination was satisfactory, the tests were confined to growing a 150 mg./cc. suspension of BCG in Corper's egg medium and Sauton's liquid medium. The same concentration was used to test virulence and allergizing potency in guinea pigs. Allowing 0.2 cc. for wastage, the contents of one ampule were divided into two doses, one-half injected intradermally into the right thigh, and one-half subcutaneously into the left thigh, so that a total of 120 mg. were introduced into each

guinea pig. Tuberculin tests with .005 mg. PPD were performed before inoculation and four weeks afterwards. Subsequent tests for allergy were made at periodic intervals.

Guinea pigs that died were necropsied carefully, and smears, cultures, and pathological sections were made from the organs to determine the presence of acid-fast bacilli and lesions that could be attributed to tuberculosis.

Technique of Vaccination

In our first trials with BCG we used the dilution indicated on the label of the ampule of dry vaccine, which was 2 cc. of sterile distilled water per ampule, or 75 mg. per cc. We made six to eight scratches on a fairly large area (3 cm. square) on the thigh, allowed the vaccine to dry, and left it without any covering. This was not wet for 48 hours. Of 33 children thus vaccinated, only three became allergic after two months.

This prompted us to use a more concentrated solution (150 mg. per cc.), and this time we adopted Rosenthal's multiple puncture technique[8] with a slight modification. By Rosenthal's method, the vaccine is allowed to dry on the arm. In our experience it took some minutes before the vaccine could dry, and for small babies who had to be carried or controlled, much time was wasted between vaccinations. We therefore thought of covering the vaccinated area with one thickness of gauze soaked with the vaccine and covering this with another square of wax paper or cellophane. The paper and gauze were plastered to the skin, to be removed in 24 hours. We advised no wetting for 48 hours. Anderson claims that covering the part is unnecessary, as there is no further penetration of vaccine almost immediately after the puncture is made.[9] However, Négre and Bretey have shown that allergy occurs earlier and more frequently where scarification is covered with a piece of cellophane for 24 hours to avoid immediate drying.[10]

The site of vaccination was usually the upper half of the arm. This was preferred to the thigh which was liable to get wet in infants.

The intradermal route was tried in 34 cases, using a concentration of 0.15 mg. in 0.1 cc., but was discontinued when 55 per cent of the 20 who returned for postvaccination testing did not react after one month. The local nodules were mostly less than 5 mm. in diameter.

The only other group on which we tried the intradermal route was on 60 children given BCG killed by heat to determine infratuberculin allergy. This work will be reported in a subsequent paper.

The skin reactions obtained after BCG vaccination were no different from those observed by Rosenthal and other workers

FIG. 2- GRAPHIC PRESENTATION OF TUBERCULIN TESTS PERFORMED IN DIFFERENT GROUPS, BY AGES, 1948

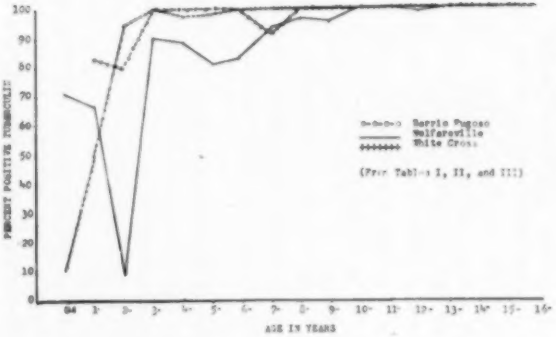
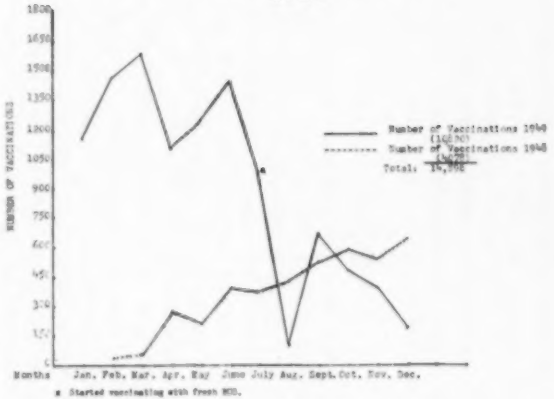


FIG. II- VACCINATIONS WITH DRY BCG OF PASTEUR INSTITUTE 1948-1949



abroad. Erythema and papule formation were the rule, sometimes progressing to nodule formation of about 2 mm. Quite rarely the punctures showed pin-point pustules of short duration. Infants below six months did not usually have as prominent "takes" as older children. Erythema was sometimes present after 24 to 48 hours, but this was usually of traumatic origin disappearing on the fourth day or earlier. Papules were observed as early as the seventh day. Papules and nodules begun to regress before the eighth week, sometimes with some exfoliation of the skin, and at the sixth month no traces could be seen, or at most only a whitish discoloration of the skin at the site of the punctures. The local reactions were no different in the revaccinated, except that they usually appeared much earlier (24 to 48 hours) and with more intensity. Groups with known contacts showed no difference in their vaccinal reactions from those with no known contacts, except that most of the cases of pustule formation took place in the contact group.

The regional lymph nodes were seldom involved. When they were, these were usually so small as to remain unnoticed, and gave no discomfort to the child. Only one case of suppurating axillary gland was observed. It was incised by a private physician, and when brought to the clinic was already healing. No acid-fast bacilli were seen on smears.

In no instance was fever or any other systemic discomfort attributed to BCG. Nor did it interfere with the child's growth and development.

Results of Tuberculin Tests

The results of the tuberculin surveys in schools and institutions are shown in Tables I to V and in Figure 1. They show a high degree of tuberculous infection among children in Manila. The figures for the slums of Barrio Fugoso (Table I) are particularly impressive. Only one child above three years, in a group of 98, was tuberculin negative. This is contrasted with the results in the White Cross Preventorium (Table III) where, despite the fact that the children came from tuberculous homes, early separation from tuberculous contacts had prevented infection in many of them, so that 89.2 per cent of those below one year were still non-reactors. Between the ages of one and two, 80 per cent of Barrio Fugoso children, and only 50 per cent of the White Cross children were infected. The group from Welfareville (Table II) also shows a high rate of infection. At the Balagtas and Sta. Ana Elementary Schools using only PPD I, children of six to seven years, the age when they first entered school, showed between 43 and 61 per cent infection, and at 18 years, all were tuberculin reactors. It appears

that the ages wherein BCG vaccination would be of most use are those from birth through pre-school age.

Results of Culture and Animal Tests

The dry vaccine, in concentrations of 150 mg. per cc. invariably showed heavy growth in Corper's egg medium and Sauton's liquid medium in three to four weeks. In some batches growth occurred as early as the eighth day.

Intradermal and subcutaneous injections into guinea pigs caused local abscesses and ulcerations. The abscesses from the intradermal injections suppurated earlier, but also healed earlier, usually before the fourth week, leaving a scar which in time disappeared completely. A few subcutaneous abscesses reached a diameter of 2 centimeters, remaining fluctuating for weeks. Some ruptured spontaneously within four weeks, and remained discharging even at the third month, but healing was uneventful, and like the intradermal abscesses, showed no traces after a time. In others,

TABLE I
TUBERCULIN TESTS ON RESIDENTS OF BARRIO FUGOSO, USING PPD I AND II, 1948

Age in Years	Number Tested	— — POSITIVE REACTORS — — Number	Percentage
1	12	10	83.3
2	5	4	80.0
3	5	5	100.0
4	4	4	100.0
5	5	5	100.0
6	5	5	100.0
7	11	10	90.9
8	9	9	100.0
9	6	6	100.0
10	6	6	100.0
11	7	7	100.0
12	3	3	100.0
13	11	11	100.0
14	2	2	100.0
15	1	1	100.0
16	..	..	.....
17	6	6	100.0
TOTAL	98	94	95.9

the abscesses did not rupture, but regressed eventually leaving no trace. Enlargement of the regional lymph nodes at the inguinal region was the rule, but was not appreciable in some instances.

Necropsies of guinea pigs that died did not show any pathological lesions referable to tuberculosis either by smear, culture, or pathological section. Acid-fast bacilli could be demonstrated only at the site of the inoculation of BCG. All vaccinated animals developed and reproduced normally.

Tuberculin allergy was produced by all but two of the batches

TABLE II

TUBERCULIN TESTS ON INMATES OF WELFAREVILLE, USING PPD I AND II, 1948

Age in Years	Number Tested	— — POSITIVE REACTORS — — Number	Percentage
0-1	7	5	71.4
1	9	6	66.6
2	10	1	10.0
3	10	9	90.0
4	17	15	88.2
5	43	35	81.4
6	24	20	83.3
7	70	65	92.8
8	75	72	96.0
9	70	67	95.7
10	51	51	100.0
11	66	66	100.0
12	54	53	98.1
13	72	72	100.0
14	40	40	100.0
15	44	44	100.0
16	29	29	100.0
17	31	31	100.0
18	27	27	100.0
19	19	19	100.0
20-24	16	16	100.0
25-29	1	1	100.0
30 & over	2	2	100.0
Unknown	11	10	90.9
TOTAL	798	756	94.7

(B-85, expiration date October 15, 1949 and B-74, expiration date July 15, 1949) within one month after inoculation, and remained up to the sixth month. Of six that were followed up to one year, four reverted. The intensity of allergy was usually 1 plus; 2 plus were few, and only two gave 3 plus and 4 plus, respectively.

Four batches of vaccines that had expired two months earlier were injected into guinea pigs in the usual manner, but gave no allergy after one month or after six months. Small abscesses were formed at the site of intradermal and subcutaneous injections. Three other batches that had expired a month earlier and inoculated in the usual manner, gave allergy within two weeks, but this disappeared in six months.

Intradermal injections at the back of the neck with 0.15, 0.45 and 0.60 mg. respectively were done simultaneously with the usual intradermal and subcutaneous injections in the thighs, using the later batches (November 1, 1949 to January 1, 1950). With the exception of those batches already mentioned, they all produced allergy within one month.

Tuberculin Allergy Following Vaccination

Only 8,931 (1,746 in 1948 and 7,185 in 1949) out of a total of 14,898 vaccinations performed with dry BCG (Figure II) from February, 1948, to December, 1949, were done by National Chest Center personnel in the prophylactic clinic proper, institutions,

TABLE III

TUBERCULIN TESTS ON CHILDREN OF THE WHITE CROSS PREVENTORIUM, USING PPD I AND II, 1948

Age in Years	Number Tested	— — POSITIVE REACTORS — — Number	Percentage
0-1	37	4	10.8
1	6	3	50.0
2	17	16	94.1
3	20	20	100.0
4	30	29	96.6
5	37	36	97.3
6	39	39	100.0
7	16	16	100.0
8	9	9	100.0
9	6	6	100.0
10-15	3	3	100.0
TOTAL	220	181	82.2

nurseries, and other special groups included in our surveys. The rest were performed in government and private hospitals and clinics that had been authorized by the National Chest Center to carry on this work. In July, 1949, the first batch of fresh vaccine was received from New York, and subsequent vaccinations by National Chest Center personnel were done exclusively with fresh vaccine. This explains the drop in the number of vaccinations performed with dry BCG from July to December, 1949 (Figure II).

Of this number, only 3,792 (42.4 per cent) have been tuberculin-tested and read at least once after vaccination. The 1948 percentage was 34.0 per cent against 44.5 per cent for 1949.

Tables VI and VII show the tuberculin reactions four weeks, eight weeks, and 12 weeks following BCG vaccinations, in 1948 and 1949, respectively. The per cent reactors obtained for infants below six months are lower than those for infants above six months to one year, and these together are much lower than for those above one year, which average above 90 per cent reactors after 12 weeks. For all ages, reactions within four weeks

TABLE IV
TUBERCULIN TESTS IN STA. ANA ELEMENTARY SCHOOL, USING PPD I, 1948

Age in Years	Number Tested	— — POSITIVE REACTORS — — Number	Percentage
5	1	1	100.0
6	26	12	46.1
7	117	55	47.0
8	267	182	68.2
9	329	234	71.12
10	307	221	71.9
11	258	198	76.74
12	271	205	75.6
13	217	176	81.1
14	175	144	82.2
15	171	134	78.3
16	101	83	82.17
17	40	34	85.0
18	7	7	100.0
19	2	2	100.0
20	1	1	100.0
TOTAL	2,290	1,689	73.7

after vaccination are usually less than those between four to eight weeks, which in turn are less than those obtained between eight and 12 weeks. In infants below six months, the percentage ranges between 63 to 64 per cent. The average in all age groups for 1948 and 1949 respectively are: between 0 to four weeks, 82.1 and 82.1 per cent; between five to eight weeks, 83.8 and 88.4 per cent; between nine to 12 weeks, 84.2 and 90.6 per cent; total average, 83.1 and 86.9 per cent. There is a close parallelism in the results obtained for both years.

Table VIII shows the intensity of tuberculin reactions following BCG vaccinations, and attempts a comparison of the degree of reactions six months and one year later. Not all the reactors within 12 weeks after vaccination returned in one year. On the other hand, there were those tested in one year who also were tested six months before. We have attempted to show the results in different institutions where certain batches of the dry vaccine were used. Some of the groups are small, nevertheless the figures suggest a definite trend.

Within the first three months after vaccination about two-thirds (50.0 to 83.3 per cent) gave a 1 plus tuberculin reaction. The other third (11.1 to 50 per cent) gave 2 plus reactions, and between 0 to

TABLE V

TUBERCULIN TESTS IN BALAGTAS ELEMENTARY SCHOOL, USING PPD I, 1948

Age in Years	Number Tested	— — POSITIVE REACTORS — — Number	Percentage
6	18	11	61.1
7	274	118	43.0
8	530	290	54.7
9	435	276	63.4
10	524	315	60.1
11	382	279	73.0
12	344	257	74.7
13	309	260	83.8
14	195	148	75.9
15	79	67	84.8
16	34	31	91.1
17	12	11	91.6
18	4	4	100.0
TOTAL	3,140	2,067	65.8

TABLE VI

TUBERCULIN REACTIONS AFTER VACCINATION WITH DRY BCG—1948

Age in Years	TOTAL TESTED			0 TO 4 WEEKS			5 TO 8 WEEKS			9 TO 12 WEEKS		
	No.	Plus	Pct. Plus	No.	Plus	Pct. Plus	No.	Plus	Pct. Plus	No.	Plus	Pct. Plus
0-5 mo.	327	244	74.6	100	64	64.0	201	159	79.1	26	21	80.7
6-1 yr.	21	15	71.4	7	5	71.4	13	9	69.2	1	1	100.0
1	62	58	93.5	29	26	89.6	28	27	96.4	5	5	100.0
2	48	47	97.9	27	26	96.3	19	19	100.0	2	2	100.0
3	37	33	89.2	19	17	89.4	17	15	88.2	1	1	100.0
4	21	20	95.2	10	10	100.0	10	9	90.0	1	1	100.0
5	27	27	100.0	20	20	100.0	7	7	100.0	..	..	....
6	17	16	93.7	12	12	100.0	3	3	100.0	1	..	0
7	13	13	100.0	10	10	100.0	2	2	100.0	1	1	100.0
8	8	8	100.0	5	5	100.0	3	3	100.0	..	..	....
9	7	7	100.0	4	4	100.0	3	3	100.0	..	..	....
10	3	3	100.0	2	2	100.0	1	1	100.0	..	..	....
11	1	1	100.0	..	..	....	1	1	100.0	..	..	....
12	1	1	100.0	1	1	100.0	..	..	....	..	..	....
13	1	1	100.0	..	..	....	1	1	100.0	..	..	....
14	..	..	....	..	..	....	..	..	....	..	..	....
15	..	..	....	..	..	....	..	..	....	..	..	....
16	..	..	....	..	..	....	..	..	....	..	..	....
17	1	1	100.0	..	..	....	1	1	100.0	..	..	....
TOTAL	595	495	83.1	246	202	82.1	310	260	83.8	38	32	84.2

TABLE VII
TUBERCULIN REACTIONS AFTER VACCINATION WITH DRY BCG—1949

Age in Years	TOTAL TESTED			0 TO 4 WEEKS			5 TO 8 WEEKS			9 TO 12 WEEKS		
	No.	Plus	Pct. Plus	No.	Plus	Pct. Plus	No.	Plus	Pct. Plus	No.	Plus	Pct. Plus
0-5 mo.	1158	885	76.4	396	253	63.8	600	499	83.1	162	133	82.0
6-1 yr.	201	173	86.0	56	50	89.2	79	68	86.0	66	55	83.3
1	559	513	91.7	136	122	89.7	218	194	88.9	205	197	96.0
2	316	294	93.0	88	82	93.1	138	130	94.1	90	82	91.1
3	193	176	91.1	63	59	93.6	71	63	88.7	59	54	91.5
4	146	137	93.8	48	45	93.7	63	59	93.6	35	33	94.2
5	133	127	95.4	44	43	97.8	62	58	93.5	27	26	96.3
6	89	83	92.1	29	27	93.1	44	42	95.4	16	14	87.5
7	93	87	93.5	38	36	94.7	35	34	97.1	20	17	85.0
8	77	76	98.7	30	30	100.0	30	30	100.0	17	16	94.1
9	56	55	98.2	20	20	100.0	27	26	96.3	9	9	100.0
10	40	39	97.5	13	13	100.0	15	14	93.3	12	12	100.0
11	32	31	96.8	13	12	92.3	14	14	100.0	5	5	100.0
12	22	21	95.4	4	4	100.0	15	14	93.3	3	3	100.0
13	10	10	100.0	2	2	100.0	6	6	100.0	2	2	100.0
14	10	10	100.0	4	4	100.0	4	4	100.0	2	2	100.0
15	6	6	100.0	2	2	100.0	4	4	100.0	0	0	
16	13	13	100.0	5	5	100.0	3	3	100.0	5	5	100.0
17	14	14	100.0	6	6	100.0	3	3	100.0	5	5	100.0
18	6	6	100.0	4	4	100.0	..	..	....	2	2	100.0
19	7	7	100.0	5	5	100.0	..	..	....	2	2	100.0
20-24	12	12	100.0	8	8	100.0	3	3	100.0	1	1	100.0
25-29	4	4	100.0	2	2	100.0	1	1	100.0	1	1	100.0
30-34	..	..	....	..	..	....	..	..	....	..	..	....
35-Over	1	1	100.0	1	1	100.0	..	..	....	..	..	....
TOTAL	3198	2780	86.9	1017	835	82.1	1435	1269	88.4	746	676	90.6

TABLE VIII — INTENSITY OF TUBERCULIN REACTIONS SIX MONTHS AND ONE YEAR FOLLOWING PREVIOUSLY POSITIVE POST-VACCINATION TESTS ON GROUPS VACCINATED WITH DIFFERENT BATCHES OF DRY BCG, 1948-1949

GROUP		PERIOD OF TEST	TOTAL	TUBERCULIN REACTIONS									
				1 PLUS		2 PLUS		3 PLUS		4 PLUS		NEGATIVE	
				No.	Pct.	No.	Pct.	No.	Pct.	No.	Pct.	No.	Pct.
National Chest Center (Mixed batches)	Known contact	0-3 mo.	167	85	50.9	68	40.7	12	7.1	2	1.2	..	..
		to 6 mo.	156	88	56.4	54	34.6	8	5.1	3	1.9	3	1.9
		to 1 yr.	34	25	73.5	8	23.5	1	2.9	..	..	..	..
	Unknown contact	0-3 mo.	239	164	68.6	70	29.2	4	1.6	1	0.4	..	..
		to 6 mo.	229	131	57.2	87	37.9	3	1.3	1	0.4	7	3.05
		to 1 yr.	70	48	68.5	13	18.5	3	4.2	2	2.8	4	5.7
White Cross Preventorium B-26 (4/15/48)*		0-3 mo.	24	12	50.0	12	50.0	..	..	..	..	..	..
		to 6 mo.	21	13	61.9	5	23.8	1	4.7	..	..	2	9.5
		to 1 yr.	9	1	11.1	2	22.2	..	..	..	..	6	66.6
Welfareville B-35 (6/1/48)		0-3 mo.	18	15	83.3	2	11.1	1	5.5	..	..	..	..
		to 6 mo.	18	2	11.1	2	11.1	..	..	..	..	14	77.7
		to 1 yr.	4	1	25.0	2	50.0	..	..	..	..	1	25.0
Welfareville B-38 (6/10/48)		0-3 mo.	12	10	83.3	2	16.6	..	..	..	..	..	..
		to 6 mo.	12	5	41.6	..	..	1	8.3	..	..	6	50.0
		to 1 yr.	6	3	50.0	..	..	..	..	..	..	3	50.0
Philippine Union College B-70 (7/1/49)		0-3 mo.	118	82	69.4	34	28.8	2	1.7	..	..	..	..
		to 6 mo.	98	69	70.4	23	23.4	..	..	..	..	6	6.1
		to 1 yr.	..	..	..	..	..	..	..	..	..	..	..

*Batch and expiration date of vaccine.

7.1 per cent gave combined 3 plus and 4 plus reactions with the greater percentage occurring in known contact groups.

The cases from the National Chest Center, which represent the results of vaccination with mixed batches of BCG, have been divided into two groups: those with known and those with unknown contact. In six months the contact group showed very little change, save for a slight diminution in the number of strong reactors, and a loss of allergy in 1.9 per cent. The unknown contact group, showed practically no change in the number of strong reactors, but had almost twice as many reverters as those in the known contact group. After one year, the known contact group had no further reverters, the strong reactors had diminished their reactions so that 97.1 per cent had 1 plus and 2 plus reactions. The unknown contact group however, increased its strong reactors to 7 per cent, but it likewise increased the number of reverters to 5.7 per cent.

At the White Cross, all the vaccinated had 1 plus and 2 plus reactions within 12 weeks. Only one developed a 3 plus reaction, and two reverted after six months (9.5 per cent).

The Welfareville group showed the largest percentage of reverters after six months. Even the single case of 3 plus reaction (B-35) in the first 12 weeks became negative. One case (B-38) enhanced her reaction from 1 plus to 3 plus in six months, but diminished it to 1 plus again in one year. An average of 66.6 per cent for both groups reverted after six months.

The Philippine Union College group consisted mostly of students living outside the school compound in Baesa, Caloocan (a rural district). Of the six who reverted, two (an American adolescent and an adult) lived within the compound, and the other four lived outside. Of the two strong reactors, one lived outside the compound.

Discussion

We repeat that in presenting our experiences with dry BCG in the past two years, we cannot give more than the immediate postvaccinal results. It is premature to touch on its protective value against tuberculosis as we are still collecting clinical data and making the follow-up of vaccinated cases as well as our "controls." However, we have shown, on the evidence of our tuberculin surveys, that a program of BCG vaccination is justifiable in the Philippines, and that our initiation of this preventive measure will do much to help in the formulation of a progressive integrated plan of tuberculosis control in this country such as those already being carried out in the more advanced nations of the world, and by the World Health Organization of the United Nations.

The important question that arises in analyzing our results is whether the dry BCG we have been using is a good vaccine, that is, whether the results obtained from it, judging from the vaccinal reactions, as well as the allergic reactions, approximate those obtained with other vaccines that have been proved of value.

The opinion has been expressed by some workers that the dry vaccine, which is a vaccine prepared by freeze-drying BCG in 50 per cent glucose as first adopted by Leschinskaya and Vakengut[11] of Russia loses much of its potency during the drying process, and when not refrigerated, most of the bacilli die. They claim therefore that vaccinating with dry BCG amounts practically to vaccinating with killed BCG, which has been shown to elicit tuberculin allergy of short duration, but has doubtful immunizing value.

Bretey, van Deinse and Senechal[12] found little loss of living bacilli from the process, but storing at room temperatures (10 to 20 degrees C.) gave only 60 per cent living bacilli after five months. They claimed better survival under refrigeration. While Birkhaug[13] claimed 95 per cent loss of living bacilli by freeze-drying, he found no further loss when stored at 2 to 4 degrees C. Ungar and Muggleton freeze-dried BCG in serum and gelatin. The vaccines showed very little loss when kept at room temperatures after one year. They sent preparations to Cuba from London to test the effect of transportation and storage, and found these viable after five months.[14]

It is possible that the dry BCG sent from Pasteur Institute had lost a certain degree of its potency. The fact that in our first trial at vaccination using the prescribed concentration of 75 mg. per cc. we obtained only three reactors out of 33 despite a large area of scarification, proves it. Adjustment to double this concentration (an allowance of 50 per cent loss) resulted in reactions in all within two months. Of these only two reverted in six months (Table VIII). The laboratory tests also prove that we were dealing with a live vaccine. The limitations under which we labored at the start of this work unfortunately did not permit us to carry out as extensive and detailed a laboratory study of the vaccine as we would have wished, but certainly we were at least able to demonstrate that with the concentrations we were using for human vaccination, good growth was obtained in artificial media between the third and fourth week, and even as early as the twelfth day; that it produced tuberculin allergy in the guinea pig as early as the second week; and that, while it produced intradermal and subcutaneous abscesses and glandular enlargements expected with the inoculation of strong concentrations of BCG, there was a typical regression of these lesions so that not one of the guinea pigs that died demonstrated any lesions

ascribable to tuberculosis, but died of other intercurrent infections.

We have also shown how vaccines that had expired only a month earlier, could still elicit allergy, and Dr. Joaquin S. Sumpaico, who heads the BCG Vaccine Laboratory, and who now conducts all tests on BCG vaccines, has obtained good growth in glycerine-water-potato medium from vaccines that had expired a month before.

Although there is conflicting evidence in the literature as to whether or not allergy is a factor in acquired resistance, it is generally accepted from practical experience, and for lack of a better indication, that when tuberculin allergy sets in, the mechanism of immunity is established. Only when a person shows a reaction following BCG vaccination can one assume therefore that it has been successful.

We have already shown how the percentage of reactors following vaccination with dry BCG gave a total average of 83.1 per cent in 1948 and 86.9 per cent in 1949. Mande and Drouhet made comparative studies between fresh and dried BCG using the scarification method.[15] They obtained 100 per cent reactors after seven weeks with the fresh BCG and 86.6 per cent with dry BCG. There is a close similarity between their results and ours. Our results still show, however, lower figures than those of Rosenthal,[8] Negre and Bretey,[10] Birkhaug,[16] Balteanu et al.,[17] and Boe,[18] but are superior to those obtained by Lind,[19] Forssman,[20] de Assis,[21] Sayago,[4] and Stransky et al.[22] The above cited utilized the multiple puncture technique using fresh vaccines, except Stransky et al., who used dry BCG. Our results are comparable with the allergy obtained after oral vaccination as reported by Kostic-Yocsik,[23] de Assis,[24] and Domingo.[25]

We have pointed out the small number of reactors in infants below one year, particularly within the first month after vaccination. The observation that infants react less to tuberculin than older children has been made by Wallgren[26] and Aronson,[27] Hertzberg,[28] Sayago,[4] and other workers. Stransky cites a personal communication from van Deinse, of the Pasteur Institute, who attributes this phenomenon to possible vitamin C defiency, or iron deficiency. We are more inclined to favor the view that there is a natively deficient antibody-forming mechanism among infants, with a consequent retardation of allergic processes.[29] In our experience we cannot overlook the fact that a significant number of vaccinated infants are brought for postvaccination tuberculin testing within the first month and do not return within the next two months. It is quite possible that many of our non-reactors subsequently became reactors during that time.

We have shown the variability in intensity of allergy six months

and one year following vaccinations, and how loss of allergy was quite marked in the Welfareville groups using two different batches of vaccine, while in the mixed batch group, and the Philippine Union College group loss of allergy occurred in less than 7 per cent. It is not improbable that different batches of vaccine may differ in potency due to some variability in the preparation, or factors of storage or temperature previous to our receiving them. Dr. Negre of the Pasteur Institute claims that in his 30 years experience, the strains of BCG kept there since 1908 show the same characteristics today as they did 30 years ago.[30] The factor of strain difference presumed by Jacox and Meade[31] who observed a similar variability as ours with regard to post-vaccination allergy, but using fresh vaccine, may not apply, therefore, to the dry BCG.

One may bring up the factor of contact to account for less reverters in the mixed batch group and the school group. We have mentioned, however, that the White Cross group is the one where least contact could possibly occur. We have made x-ray surveys with our mobile unit in all these institutions, and found the White Cross and the Philippine Union College to have comparatively lower percentages of suspected or definite tuberculosis. At Welfareville many more had active and inactive forms of tuberculosis. One would therefore have expected more cases of superinfection in the Welfareville group and by inference, less reverters, which was not the case.

Table VIII also supports our contention that a so-called non-contact group in the Philippines would be impossible to find. The unknown contact group showed less number of strong reactors, and almost twice as many reverters than the contact group after six months, but in one year there were two and one half times more strong reactors. It is generally assumed that a sudden increase in intensity of the tuberculin reaction denotes the existence of a superinfection with virulent tubercle bacilli even in the absence of clinical symptoms.

The question also arises whether the reversion that followed the previously positive tuberculin test is an indication of a true loss of allergy (and consequently of immunity) or simply a latent or "infratuberculinic" state as de Assis first called it.[32] All our re-vaccinations on the White Cross and Welfareville children, with one exception, elicited the so-called "accelerated" reaction or Willis-Sayé phenomenon also known as the Baldwin-Gardner-Willis-Sayé phenomenon,[33] showing that allergy remained latent. Some investigators consider that this shows that acquired resistance may outlast allergy.

The one exception mentioned, and two others in our experience,

did not react to tuberculin despite accelerated vaccinal reactions following two revaccinations, finally giving a 1 plus reaction after some time. These persistently negative cases may be among those referred to by Sayago,[34] Kereszturi, et al.,[35] and Wallgren as being constitutionally unable to develop a general sensitivity of the body even with highest doses of tuberculin. We tried injecting 1 mg. O.T. in these children and in one of them only a faint erythema was produced. Holm[36] states that occasionally such persons will have a sensitivity that endures only a very short time.

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SUMMARY

1) The dry BCG vaccine of the Pasteur Institute, at concentrations of 150 mg./cc. has been found viable, innocuous, and capable of producing allergy in guinea pigs and humans after multipuncture, intradermal, and subcutaneous inoculations.

2) With dry BCG 14,898 vaccinations were performed from February, 1948 to December, 1949, using the multipuncture technique. Of 8,931 performed by National Chest Center personnel, 3,792 or 42.4 per cent returned at least once for postvaccination tuberculin testing.

3) Postvaccination tuberculin allergy occurred in 83.1 to 86.9 per cent for all age-groups, within 12 weeks.

4) Infants below one year gave a lower percentage of reactors and poorer skin reactions.

5) The intensity of allergy after vaccination is usually of a weak character, and has a tendency to attenuate.

6) The duration of allergy varies with different batches of vaccine, but in general, more than 80 per cent remain allergic after six months and one year. The procedure of tuberculin testing all vaccinated individuals every six months is recommended.

7) In the light of the high degree of tuberculous infection taking place among Filipino children, BCG vaccination is justifiable as

a supplementary measure in the control of tuberculosis in the Philippines.

RESUMEN

1) La vacuna BCG seca del Instituto Pasteur a concentraciones de 150 mg./cc. se ha encontrado viable, inocua y capaz de producir alergia en cobayos y seres humanos después de multipuntura, inoculación intradérmica y subcutánea.

2) Se han realizado 14,898 vacunaciones con BCG seco de Febrero de 1948 a Diciembre de 1949, usando la técnica de multipuntura. De 8,931 practicadas en el Centro Nacional de Enfermedades del Tórax 3,792 o sea el 42.4 por ciento regresaron por lo menos una vez para prueba tuberculínica postvacunal.

3) La alergia postvacunal a la tuberculina ocurrió en 81.1 por ciento a 86.9 por ciento en todos los grupos dentro de 12 semanas.

4) Los infantes bajo un año de edad dieron un porcentaje menor de reactores y más pobres reacciones cutáneas.

5) La intensidad de la alergia después de la vacunación es generalmente escasa y con tendencia a atenuarse.

6) La duración de la alergia varía con los diferentes lotes de vacuna, pero en general más del 80 por ciento permanecen alérgicos después de seis meses y aún de un año. Se recomienda el procedimiento de vacunar a todos los vacunados cada seis meses.

7) Ante el alto grado de infección tuberculosa de los nikos filipinos, la vacunación por el BCG está justificada como medida suplementaria en el control de la tuberculosis en las Filipinas.

RESUME

1) Le BCG sec de l'Institut Pasteur à la concentration de 150 mmg par CC s'est démontré être actif, dépourvu de nocivité et susceptible de conférer l'allergie chez le cobaye et l'homme par voie intradermique ou cutanée.

2) De Février 1948 à Décembre 1949, 14,898 vaccinations ont été faites par le BCG sec. La technique utilisée fut celle des "piqures multiples." Sur les 8,931 individus vaccinés par le personnel du Centre National des Affections Thoraciques, 3,792, soit 42.4%, revinrent au moins une fois pour le contrôle de leur allergie post vaccinale.

3) Cette allergie apparut en 12 semaines chez 83.1 à 86.9 pour cent des vaccinés appartenant à toutes les catégories d'âge.

4) Chez les nourrissons de moins d'un an on constata un pourcentage moindre de ráaction positive.

5) L'allergie obtenue par la vaccination est habituellement faible et a tendance à s'atténuer.

6) La persistance de l'allergie varie selon les différents lots de

vaccin mais en général plus de 80% des sujets restent allergiques après 6 mois et 1 an. Les auteurs recommandent de vérifier les réactions tuberculiniques des vaccinés tous les six mois.

7) Etant donné l'importance de l'infection tuberculeuse chez les enfants aux Philippines, il est légitime de faire appel au BCG comme mesure complémentaire dans la lutte contre la tuberculose.

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The Role of Para-Aminosalicylic Acid (PAS) in the Emergence of Streptomycin-Resistant Tubercle Bacilli*

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Clinical and laboratory experimentation has shown the importance of drug resistance of the tubercle bacilli to streptomycin in the treatment of tuberculous patients.[1,2] Streptomycin is now being used by many physicians as the sole therapeutic agent in tuberculosis, disregarding the importance of drug resistance (in vitro). It is the authors' feeling that the use of streptomycin, if given without PAS, should be limited to those patients whose tubercle bacilli are found to be sensitive and which remain so during treatment. Among 21 patients of this hospital on whom retreatment with streptomycin was attempted in spite of the presence of resistance to this antimicrobiotic, favorable clinical response was encountered in only one case, and even in this one there was no convincing radiological evidence of improvement. A new approach to this problem has been offered to us by participation in the Veterans Administration-Army-Navy Research Program, which has used PAS in combination with streptomycin and we feel that our data may add to the overall picture in the treatment of tuberculosis by combined therapy. Lehmann has demonstrated that PAS is effective in vitro on the tubercle bacilli and is also active in vivo.[3,4] Further work on the combined effect of streptomycin and PAS in experimental tuberculosis in mice by Youmans, Department of Bacteriology, Northwestern Medical School, has shown the additive or potentiating effect with combined use of these drugs.

In our work it has been kept in mind that by lowering the total

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dosage of streptomycin and by adding a synergistic drug, the emergence of streptomycin resistance may be delayed. Another factor which has been considered in our study is that, by lowering the total dosage of either drug, the toxicity may be lowered, since the toxicity of any drug is directly related to its total dosage.

Procedures and Methods

The following resistance study protocols have been incorporated in this paper for evaluation:

Protocol 1. Streptomycin 1 gm., daily for 42 days.
Protocol 2. Streptomycin 1 gm. daily, PAS 8 gms., daily for 120 days.
Protocol 3. Streptomycin 1 gm. daily, PAS 15 gms., daily for 120 days.
Protocol 4. Streptomycin 1 gm. daily, PAS 12 gms., daily for 90 days.
Protocol 5. Streptomycin 1 gm. daily, PAS 12 gms., daily for 120 days.

Streptomycin sensitivity tests were performed prior to treatment and at regular intervals thereafter. The sensitivities were recorded at 60 days, 90 days and 120 days after starting treatment. All pre-streptomycin cases were found to have the great majority of their tubercle bacilli sensitive to less than three micrograms of streptomycin per cc. A patient was not recorded as resistant until at least a 2+ growth was observed on the 10 mcgm./cc. tube.

Streptomycin sensitivity determinations were conducted on each sputum at O concentration (control), 3, 10 and 100 mcgm./cc. of

FIGURE 1

Comparative Resistance Studies

JULY 15, 1950	Total No. Tested	— 60 DAYS —			— 90 DAYS —			— 120 DAYS —		
Resistance Studies		Number Tested	No. Resistant to $\wedge$ 10 mcgm.	Per cent Resistant	Number Tested	No. Resistant to $\wedge$ 10 mcgm.	Per cent Resistant	Number Tested	No. Resistant to $\wedge$ 10 mcgm.	Per cent Resistant
SM 1 - 42 D.	43	29	11	37	19	9	47	22	12	54
SM 1, PAS 8, 120 D.	23	22	4	18	21	4	19	17	2	12
SM 1, PAS 15, 120 D.	29	29	3	10	27	2	07	23	2	08
SM 1, PAS 12, 90 D.	29	28	5	21	23	3	13	22	3	14
SM 1, PAS 12, 120 D.	38	38	1	03	27	2	08	26	1	04

medium. The solid media used were Jensen-Lowenstein and/or A.T.S.[5] The method of reporting is listed below:

4+: Surface of medium entirely covered.
3+: Growth not quite confluent.
2+: Innumerable colonies.
1+: 50 to 200 colonies.

Actual colony counts were made if less than fifty were observed.

Comments

The reduction in the number of patients tested at 120 days as compared with 60 days, reflects the marked reduction in positive sputa during treatment. On many occasions positive smears were obtained, but the culture of these specimens proved to be negative. Whether this really indicates definitive therapeutic effect or merely a suppression by the treatment with conversion at a later date, only time will tell. It does seem that the viability of the tubercle bacilli as revealed by cultural methods in many cases is lowered during actual therapy.

From the data comparing the 42 day 1 gm. streptomycin daily regimen with the streptomycin 1 gm. daily and PAS 12, 15 or 8 gm. daily regimens, it will be observed there has been a marked reduction in the number of patients developing resistance to streptomycin.

Unfortunately we have been unable to conduct PAS sensitivity determinations on all our patients at the intervals shown on our chart. In determining the sensitivities on 36 patients prior to and during therapy, four of the total treated with combined therapy have developed resistance to PAS. According to Bo Carstensen and K. Linde Andersen,[6] resistance in vitro to PAS often appears after six months, the earliest appearance being at 112 days. In contradistinction to the previously accepted view that the emergence of in vitro resistance to PAS is without practical significance, because no corresponding reduction of the effectiveness of this drug occurs in vivo, the above authors found definite correlation between in vitro resistance and clinical results. They also come to the interesting conclusion that the simultaneous use of streptomycin has no delaying effect upon the emergence of resistance to PAS.

Acknowledgment: We wish to express our sincere appreciation to Mr. T. Tewey, Streptomycin Clerk, for keeping our records and aiding in our research program.

SUMMARY

There has been a marked reduction in the number of patients showing tubercle bacilli which are resistant to streptomycin when combined therapy with PAS is employed.

RESUMEN

Ha habido una decidida reducción en el número de pacientes que muestran bacilos tuberculosos resistentes a la estreptomicina cuando se ha empleado la terapia combinada con el PAS.

RESUME

Le nombre des malades chez qui apparaissent des bacilles tuberculeux résistants à la streptomycine diminue considérablement quand on institue le traitement combiné avec le PAS.

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Psychogenic Factors in Chest Diseases*

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A few years ago if a doctor had expounded on the subject of psychogenic factors in chest diseases, we would have raised our eyebrows and wondered what had become of his scientific reasoning. Today, due to the impact of psychosomatic medicine we consider it a perfectly respectable subject. Now we know that psychosomatic medicine has brought home to us once more the healing properties of friendly understanding. If we can in our own daily practice impress upon our patients the fact that we wish to hear their stories rather than to utter commands, we will have removed a portion of the fear and reluctance that so many people experience in coming to a doctor. Hendricks says: "In the application of psychosomatic medicine we must study the patient as a human being, as well as the disease which he may have acquired. If we are to be successful in applying treatment in full measure to the tuberculous patient, we must know our patient as a member of society and come to understand his emotions and reactions to past situations and to new situations. We must recognize and study his anxieties and the background of his particular problems. We must recognize the fact that no examination is complete without a study of the personality and social adjustment of the patient." "... It is now believed by most phthisiologists and psychiatrists that the high incidence of neurosis in tuberculous individuals is not actually due to the disease itself but that these neuroses and their trends in tuberculous individuals existed prior to the onset of active tuberculosis and that the neurotic trends are accentuated by the disease."

Weiss and English say: "Psychosomatic is a relatively new term, but it describes an approach to medicine as old as the art of healing itself. It is not a specialty, but rather a point of view which applies to all aspects of medicine and surgery. It does not mean to study the soma less; it is only to study the psyche more. It is reaffirmation of the ancient principle that the mind and body are one, that they function as interacting and interdependent organs—a principle which has always guided the intelligent general practitioner."

I approach this subject, therefore, with both hope and enthu-

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siasm because I feel that if we, of the American College of Chest Physicians—or any other specialty group for that matter—took seriously to understand and practice psychosomatic medicine, we would find a greatly improved specialty. We would find a closer relationship between doctor and patient, a mutual understanding of each other's problems, a friendly attitude of one for the other. One can even picture this splendid harmonious relationship growing to that state where conceivably the patient would understand and forgive the doctor for his temper tantrums or ill-chosen words —where the patient might even say—"Doctor, I feel much more pleased with myself since meeting you." To have attained such a wholesome degree of fine teamwork between doctor and patient would indeed be a mark of distinction for any specialty.

No one knows definitely how the psyche first began. The concept voiced by psychiatrist William A. White, however, is both interesting and instructive. It gives us a sensible starting point. In the beginning the simplest organism became structuralized by virtue of repeated contacts with its environment. If one part of the organism received repeated stimuli, it soon developed structural pathways which transmitted the stimulus to other parts of the organism. The part receiving these stimuli from the environment became the head—the brain—the directing mechanism to the rest of the body.

After ages of development, the brain became a highly specialized organ. It has learned ways and means of behavior and misbehavior and had stored volumes and volumes of memories. These factors made it a very tricky organ. If we paused to consider some of the complex reactions that the brain became subjected to, none is more interesting that the subject of taboos because they, in turn, exercised a tremendously important influence on the psyche of the individual.

Frazer discusses taboos and their effect upon the primitive mind. The taboo being a danger, he considers it not less real because it is imaginary. "Imagination," he states, "acts upon man as really as does gravitation and may kill him as certainly as a dose of prussic acid."

The taboos in our modern life are the fears that make men cringe. They are the "Dont's" that we put on our patients and they should be dispensed as carefully as any other medicine. In one form or other the "Do's" and "Dont's" in the life of the individual make up to a great extent the psychosomatic pattern of the person. They result in the conflicts that besiege the soul and distress the spirit to the eventual question—"Now what shall I do?" The effect of these psychic conflicts on the body, even to the extent of producing pain and other distressing symptoms, has

resulted in one of the most interesting studies in medicine—Psychosomatic Disease.

This rather amazing phenomenon of mind effect over the body has led to many philosophical speculations. The question naturally arises—"When is it psyche influence over the some or when is it soma influence over the psyche?" That can lead to many roads of discussion. Our present paper proposes to deal with the psyche influences as they affect the reaction of the body and to show what an important part of our practise this has become.

Weiss and English devote a chapter to the respiratory system. They point out the physiological effects of the emotions on this system and cite case reports illustrating the influence of the psyche on chest diseases.

The psychogenic factors in bronchial asthma are already well known. McDermot and Cobb, in a psychiatric study of 50 cases of bronchial asthma, found that 37 of the 50 seemed to have an emotional component in the asthmatic attacks. Twenty patients reported that the first attack was emotionally precipitated.

Gillespie, of London says: "An idea may become the affective stimulus which elicits the asthmatic response just as much as pollen or horsehair."

In the individual's battle against tuberculosis there are many psychogenic factors—anxiety, fears, hatreds, and so forth—that may definitely influence recovery. On our rounds of the sick, we frequently hear such expressions as "He doesn't want to get well," "He's lost his morale," "She's quit fighting," or "She's scared to death." What do these expressions signify? To me they mean that people generally are cognizant of the fact that the psyche has a great influence over physical processes and that when one loses morale, then the fight is lost. This general belief is supported by medical evidence, but we have been slow to learn how deep the relationship goes. For all we know psychic insults may hit the individual cell a much greater blow than we realize.

Back in 1926, and of course even earlier, the psychosomatic factor in tuberculosis was recognized but not fully appreciated. At that time, however, White cites this interesting case: "A young woman has a very ardent love affair which, however, comes to naught. The engagement is broken off. Her heart is broken; she becomes depressed, loses her appetite, ceases her social activities, gives up her interests, becomes more and more self-centered, loses flesh, has insomnia, and develops pulmonary tuberculosis. Treatment addressed to the psychological situation, which restores her hope and brings back her interests, results in cure not only of the mental depression but of the tuberculosis."

To stress further this point, I wish to mention the case of a young man who had had the best type of sanatorium care for his bilateral pulmonary tuberculosis. Despite this, however, he continued to throw his meals, had night sweats and fatigue out of all proportion to his lesion, was depressed, fearful, and resigned to the worst.

His emotional lesion was so much more in evidence than his tuberculous lesion that he was informed to forget about his tuberculosis. It seemed like a violation of every good therapeutic principle in tuberculosis treatment to tell this young man to get up, get out, and get interested in some outside activity. Nevertheless, it was done and much to my surprise and delight, the weeks brought emotional readjustment and a return of appetite and strength. The pulmonary lesion continued to improve. Actually the boy was caught playing basketball. When I learned of that, I concluded that with all due respect to the psyche, we should not push the soma too far—so he gave up basketball.

I do not wish to leave the impression that we were so concerned with this young man's psychic lesion that we forgot about the tuberculous lesion. Periodic checks were made by x-ray films and sedimentation rates to detect pathologic changes. We reduced these to a minimum, however, to every three months and then six months. Why? Because from past examinations the tuberculous lesion had shown a high degree of stability and also because each x-ray film and laboratory examination in this particular individual evoked a severe emotional upset. It was felt that with this young man the greatest value of his doctors visits consisted in a sympathetic appraisal of his fears, an explanation of how fears can produce symptoms similar to organic disease, and frequent reassurance regarding the stability of his tuberculous lesion.

The above ministrations, plus gentle guiding, will help many tuberculous patients to gain a better philosophy of life and consequently a happier state of mind and body than would occur without the doctor's sympathetic understanding. Thus it becomes of increasing importance in the practice of chest diseases to apply to the fullest and wisest degree the principles of psychosomatic therapy to our patients.

The effect of the emotions on increasing the output of adrenaline is familiar to us all. I was not impressed, however, until recently that emotions may also have a pronounced effect upon bronchial secretion. A very interesting study of this was made by Stevenson and Wolff. They concluded as follows: "In a subject with bronchiectasis, it was found that during stressful life situations the bronchial mucus secretions increased markedly, sometimes as much as eightfold, and again diminished when the stress

was over." Many other studies have helped to confirm the fact that psychogenic factors are at work in chest diseases.

There is a final psychogenic factor that affects the patient and that is the physician himself. I doubt if we emphasize this factor sufficiently. It is a potent one and may win or lose the case. Hendricks says: "The physician who hopes to be successful in the management and treatment of tuberculous patients must be one of great understanding. He should like people and be interested in their welfare and happiness. He must be able to advise them on many problems, nonmedical as well as medical." For the best psychosomatic medicine, one must have the ideal physician. He must be a man of steel, immune to fatigue and with a charming personality. He must have to the fullest degree patience, poise and love of life. He must be rich in medical science, diplomacy, understanding and goodness; full of knowledge, wisdom and intelligence. He must have a sense of humor which misses no witicism and produces many. He must have all the time in the world so he hurries on one and no one hurries him. He must have the wisdom to place first things first and have the details fall into their proper places. He must have a general diagnostic sense with a keen eye to separate the functional from the organic. All these things—and more—he must have to be the ideal psychosomatist.

Of course none of us can fulfill all of these requirements. We can, however, be conscious of these virtues and recognize their value in the better treatment of the patient. The threapeutic value of the physician himself is great and can be a wonderful force if properly directed, as Houston has so wisely stated in "The Art of Treatment": "If a physician learns to know himself, he will see that he himself is a potential neurasthenic or psychasthenic. The outcome for the individual will depend on the toughness of his constitutional fiber (just how tough it is can scarcely be known until it is put to test) and upon the environmental conditions. Among the environmental conditions to which the patient is exposed is the doctor. As an environmental factor, the doctor is sometimes an unfortunate one, but in the favorable case it may be the greatest good fortune that could befall the patient to have come into contact with the doctor." The need of an understanding physician is of paramount importance in the treatment of a tuberculous patient because fear of the disease and fear of its consequences are, in most cases, a most disturbing factor. Houston further states: "In its extremest forms, the very agony of fear, fear is the most powerful of the emotions. The visceral changes that accompany such fear are of extreme violence. The heart bounds, halts, then races; the eyes start and dilate; the skin is pale and sweats; the hair stands on end; control of the sphincters

may be lost. Such a picture of abject terror may be seen in a young recruit first under fire, but it is to be observed to a minor degree every day in patients. They are afraid of having the mysteries of their bodies explored; they fear some ominous verdict; they fear that they will be hurt; they most often consult a doctor because they are in a state of fear. Frequently they could endure with equanimity the discomforts that they suffer; it is the fear of what the discomforts may portend that leads them to the doctor."

The question naturally arises, which nervous patients can we in our field handle and which should we send to the psychiatrist. We cannot lay down any hard and fast rules in this regard because some chest physicians are better than others both by nature and training to treat the emotional patient. In this regard we must use our diagnostic acumen. We must learn to select those cases that need more psychiatric help than our training permits us to give. By and large, however, we will find that most of the patients we encounter are not in need of specialized help in their emotional struggles, but can be cared for by good sensible, counseling by an understanding physician.

SUMMARY

Without enlarging further upon the need for psychosomatic treatment for our chest patients, let us consider briefly the value of this step to our specialty. If every chest physician made a special effort to deal with his patient not only as a case of organic disease, but as an emotionally sick, fearful and anxious individual, he would find such a grateful response as would indeed warm his heart. The widespread practice of this art would soon label the chest physician as outstanding in his understanding of the care of the sick individual. Could we ask for a finer compliment?

Let me implore that we hold fast to those agents, in the combating of tuberculosis, that have passed the test of time and science. We are today emphasizing the psychosomatic angle in order that we may add to the effectiveness of our therapeutic agents and not detract from their value.

RESUMEN

Sin ir más lejos sobre la necesidad de la terapia psicosomática en los enfermos del tórax, consideramos brevemente el valor de esto en nuestra especialidad. Si todo especialista de tórax hiciese un esfuerzo especial para tratar a sus enfermos no solo como un caso de enfermedad orgánica sino como un enfermo emocional, temeroso y lleno de ansiedad, encontraría una grata sorpresa que lo alentaría.

La práctica más extensa de este arte sin duda que haría desta-

car al tisiólogo por su cuidado de los enfermos. Podríamos pedir mayor cumplido?

Dejadme implorar que nos atengamos a aquellos agentes para combatir la tuberculosis que han pasado la prueba del tiempo y de la ciencia. Ahora estamos enfatizando el aspecto psicosomático para que lo podamos agregar a la efectividad de nuestra terapéutica y no se desprecie su valor.

RESUME

Sans développer outre mesure l'utilité du traitement psychosomatique pour nos malades atteints d'affections thoraciques, considérons rapidement la valeur de cette acquisition dans notre spécialité. Si tout médecin des voies respiratoires faisait un effort pour ne pas voir seulement chez son patient un cas d'affection organique, mais aussi un individu atteint dans son émotivité, craintif et anxieux, il trouverait en retour une gratitude émouvante. Ainsi le spécialiste serait vite considéré comme un médecin complet dans la compréhension des soins à donner au malade. Peut-on demander un plus beau compliment?

Laissez-moi demander que nous attachions avec attention aux éléments qui dans la lutte contre la tuberculose ont dépassé l'expérience du temps et de la science. Nous insistons aujourd'hui sur le point de vue psychosomatique pour l'adjoindre à l'efficacité de nos moyens thérapeutiques et non pour dénier leur valeur.

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Classification of Pulmonary Tuberculosis with Five Digit Code

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Many attempts have been made in the past to devise an accurate classification of the various types of pulmonary tuberculosis. It is particularly difficult for a classification to meet all the requirements of clinicians, anti-tuberculosis institutions, workers in vital statistics as well as those working in mass examinations of millions of apparently healthy people.

Amongst all available classifications the best is the one compiled by Albrecht-Fraenkel later modified by Aschoff-Nicol. The last classification of the National Tuberculosis Association of America (1940) comprises the basic elements of the Aschoff-Nicol classification, such as the pathologic-anatomic form and clinical status of the disease. However, it has become apparent that this classification needs revision because it cannot comprehend certain types of pulmonary tuberculosis and give information as to relative infectivity.

To meet the urgent demand for a universally acceptable system of classifying tuberculous disease of the lungs, Hilleboe and Holm[1] devised a purely descriptive classification, which can be summarised in a simple four digit code. Ten selected findings for each of four categories have been arranged in a pattern. Each finding is assigned a digit form from 0 to 9 and as the order of the digit increases, the finding increases in seriousness. The code then is a four digit number, with the digit positioned so that the first digit always relates to the first category. (T) tubercle bacilli, the second to (C) cavities, the third to (L) lesions and the fourth to (F) principal symptoms.

Comparison of four digit code for the same individual examined at different times, readily reveals progress of the disease, when the latest code contains digits that are higher than before. A patient's improvement is reflected by a code with digits of a lower order.

The classification of Hilleboe and Holm unquestionably gives accurate information as to relative infectivity and diagnosis of cavities and of principal symptoms. It is however, at a disadvantage since it gives no information as to the type of tuberculosis

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(primary and reinfection), for the various forms of the disease and for the clinical status.

These disadvantages urged us to devise a descriptive classification of pulmonary tuberculosis and to summarize in a five digit code. This classification should comprise all the advantages of the Hilleboe and Holm classification and give in addition accurate information of the type, various forms and clinical status of the disease in connection with the symptomatology.

In our classification the code is summarized in a five digit number, with the digit positioned so that the first digit always relates to the first category (T) tubercle bacilli, the second to the type and form (F) of the disease, the third to the localization of lesions (L), the fourth to the cavities and pleural exudate (C) and the fifth to the clinical status in connection with the symptomatology (S). Ten selected findings for each of the five categories are arranged in a pattern. Each finding is assigned a digit from 0 to 9 and as the order of the digit increases, the finding increases in seriousness. Special care has been taken so that the findings of the classification shall be objective. Thus the classification makes possible the accurate diagnosis of the disease and in addition gives indication as to the prognosis and treatment.

The findings for the detection of the tubercle bacilli are based on the modern microbiological methods of examination, viz., the direct smear examination, the sputum concentrate, the sputum culture and gastric or bronchial lavage culture (Table I).

The microbiological examination must begin by the most simple procedure of direct smear microscopy and go on progressively to the above mentioned methods of detection in case of negative results.

The diagnosis of type and form of pulmonary tuberculosis is made by x-ray inspection. The interpretation of pathologic

TABLE I

LABORATORY

Tubercle Bacilli: Present +, Absent 0

0	No examination	—
1	Gastric (or Bronchial) Lavage Culture	0
2	Sputum Culture	0
3	Sputum concentrate (no culture made)	0
4	Sputum. Direct Smear (no other examination made)	0
5	No expectoration. Gastric (or bronchial) lavage not done	—
6	Gastric (or bronchial) Lavage Culture	+
7	Sputum Culture	+
8	Sputum concentrate	+
9	Sputum Direct Smear	+

anatomic form of the disease—productive, exudative and mixed forms—from x-ray films, presents certain difficulties. According to Hilleboe and Holm, specialists in tuberculosis in the United States have been in disagreement concerning from 33 to 50 per cent of the chest films which were read. Therefore, in our classification, the x-ray findings of pulmonary tuberculosis are divided in two categories, a) primary tuberculosis and b) reinfection tuberculosis, endogenous or exogenous.

As for the primary tuberculosis, as is well known, we have two forms, a) the perihilar infiltrate described by Slucka and b) the fissular infiltrate with or without interlobar exudation (Table II).

Concerning reinfection tuberculosis, we follow the differentiation based on morphological elements interpreted from the chest plates, e.g., nodular, parenchymal inflammatory alterations and mixed forms.

The nodular form is divided in three sub-divisions, a) the form of early infiltrate, b) of nodular dissemination and c) of the generalized nodular (miliary). The parenchymal inflammatory alteration forms are subdivided in the a) form showing homogeneous patch and b) nonhomogeneous patch. Finally for the mixed form we adopted the mixed form a) with prevailing nodular alterations and b) with prevailing patchy infiltrations.

This classification of the x-ray findings of pulmonary tuberculosis satisfies our modern scientific demands and in addition has the advantage of easy interpretation by all tuberculosis specialists. In cases where pneumothorax or thoracoplasty is applied, when the reading of the forms and cavities is impaired, the number of digit No. 2 is replaced by Pn for Pneumothorax and Th for Thoracoplasty.

TABLE II

CHEST X-RAY FILM INSPECTION

Type and forms of Tuberculosis

0	No alterations in the lung fields.
1	Primary tuberculosis perihilar infiltrate.
2	Primary tuberculosis fissural infiltrate.
3	Reinfection tuberculosis early infiltrate.
4	Reinfection tuberculosis nodular dissemination.
5	Reinfection tuberculosis generalized nodular (miliary).
6	Reinfection tuberculosis mixed, prevailing nodular.
7	Reinfection tuberculosis mixed, prevailing patch.
8	Reinfection tuberculosis homogeneous patch.
9	Reinfection tuberculosis nonhomogeneous patch.

(Pn) Pneumothorax or (Th) Thoracoplasty obscures field preventing cavity and lesion identification, enter Pn or Th instead of digit.

Table III of the code contains the localization of the lesions of the various forms. The lung is divided into apex, upper lung and lower lung, according to the classification of Hilleboe and Holm. The apex comprises the area of lung field above the fourth rib posteriorly, the upper lung includes apex and two interspaces below the fourth rib posterirly and the lower lung the interspaces below the sixth rib posteriorly.

Table III covers all possible combinations with the exception that numbers 1, 3 and 8 must be accompanied by the letters (U) for upper lung and (L) for lower lung. In order to determine which lung is concerned we add before the number (R) for right side and (L) for left side, the differentiation always concerning the apex or upper lung. For example I over R U stands for right upper lung.

After the application of pneumothorax or plastic operation the number of the digit No. 3 indicating localizations remains the same. Eventual extension of the disease is formulated by altering the number of the digit No. 3 according to the extension involved.

Table IV refers to cavities and pleural effusions. Pleural effusions have been comprised in the classification, because they may be due to pleurisy with or without tuberculosis of the lung or due to pneumothorax complication.

Our classification includes also cavities because of their importance for diagnosis, prognosis and treatment. As cavities can exist in one or both sides or can be only suspected, Table IV covers all possible combinations of cavities and pleural effusions.

Where pneumothorax or thoracoplasty is applied, causing im-

TABLE III

CHEST X-RAY FILM INSPECTION

Localization of Lesions

0	Apex of one lung.
1	Upper or lower lung of one side.
2	Apices of both lungs.
3	Apex with upper or lower lung of the other side.
4	One lung.
5	Both upper lungs.
6	Upper lung with lower lung of the other side.
7	Both lower lungs.
8	Apex or upper or lower lung with the other lung.
9	Both lungs.

Apex: Area of lung field above fourth rib posteriorly.
U: Upper lung including apex and two interspaces below fourth rib posteriorly.
R: Right lung. L: Left lung.

pairment of the reading of cavities, the digit indicating cavity is replaced by digit indicating suspected cavity.

Finally Table V contains findings concerning the symptomatology and the clinical status of the disease. The patient should be a reactor to tuberculin. The only exceptions will be persons acutely ill or in the final stages of the disease since the allergic reaction to tuberculin is often absent in such persons. Blood sedimentation rates can contribute to the diagnosis and give indication as to the prognosis of the disease where they are not due to any other pathological cause. The principal symptoms are fever, tachycardia, dyspnea, sputum, cough, weight loss, fatigue, sweating. The physician's judgement must be the criterion of their severity. The following definitions are necessarily approximate:

TABLE IV

CHEST X-RAY FILM INSPECTION

Cavities and Pleural Effusions

0	No cavity.
1	Effusion in pleural space.
2	Suspected cavity on one side.
3	Effusion in pleural space with suspected cavity on one side.
4	Suspected cavities on both sides.
5	Effusion in pleural space with suspected cavities on both sides.
6	Cavity on one side.
7	Effusion in pleural space and cavity on one side.
8	Cavities on both sides.
9	Effusion in pleural space with cavities on both sides.

TABLE V

CLINICAL STATUS: SYMPTOMATOLOGY

0	Clinically not significant (apparently healed more than 2 years ago).
1	Clinically not significant (apparently healed less than 2 years ago.
2	Probably clinically significant. Observation case without symptoms.
3	Probably clinically significant. Observation case with slight symptoms.
4	Clinically significant. Treatment case without symptoms.
5	Clinically significant. Treatment case with slight symptoms.
6	Clinically significant. Treatment case with moderate symptoms.
7	Clinically significant. Treatment case with severe symptoms.
8	Clinically significant. Treatment case with moderate symptoms and extrapulmonary tuberculosis.
9	Clinically significant. Treatment case with severe symptoms and extrapulmonary tuberculosis.

Testing: Reaction to old tuberculin or P.P.D. necessary, except for terminal cases. Blood sedimentation rate.

Principal symptoms: Temperature, tachycardia, weight loss, cough, fatigue, sweats.

None, slight, moderate and severe. In moderate and severe cases, there must clearly exist feverish conditions.

As far as the clinical status is concerned, these are subdivided into a) clinically non-significant, b) clinically probably significant and c) clinically significant.

The clinically non-significant cases present no toxic symptoms and the x-ray film shows fibrotic or fibro-calcified lesions. These again are subdivided into cases apparently healed more than two years ago, and to those healed less than two years ago.

As clinically probably significant cases, we considered observation cases, which at most can only present slight symptoms and which on x-ray inspection show productive lesions tending to fibrosis.

The clinically significant ones are considered the treatment cases, the symptoms of which may vary according to the resistance of individual subjects. No. 8 and 9 in Table V comprise also treatment cases with moderate and severe symptoms with extrapulmonary tuberculosis.

Table V covers all possible combinations of clinical status correlated to symptoms.

An analysis of our Tables and their comparison with the tables of Hilleboe and Holm code system demonstrates that our code system is more complete in that it contains type and forms as well as the clinical status of the disease. In addition, we believe it to be more simple and handier in use. Moreover, as it fulfills our present scientific requirements we propose it for universal use.

Below we give examples describing status and evolution of the disease by our code system.

Case 1, 2.1.49/15907: Miliary tuberculosis. Negative gastric lavage culture, generalized nodular infiltrations in both lungs, clinically significant with severe symptoms.

Case 2, 30.9.48/14200: Healed lung tuberculosis, with negative gastric lavage culture, disseminated fibroid nodules in both apices, with no symptoms.

Case 2, 25.1.49/97226: Relapses of this case positive by sputum direct smear examination, in both apices mixed prevailing patch infiltrations with cavity on one side and moderate symptoms.

Case 3, 10.8.48/10117/R L: Pleurisy of one side. Negative gastric lavage culture, no alterations in the lung fields, effusion in the right pleura with severe symptoms (high temperature, tachycardia, dyspnoea, etc.).

Case 3, 12.11.48/10111/R L: Absorption of fluid, formation of adhesions, negative gastric lavage culture, recently apparently healed with no symptoms.

Case 4, 28.1.48/13104/R U: Primary infiltration with negative gastric lavage culture, in the right upper lung with no cavity and no symptoms.

Case 4, 2.2.48/83166/R U: Worsening, positive sputum concentrate, formation of cavity with moderate symptoms.

Case 4, 21.5.48/1Ph124/R U: Application of pneumothorax, improvement, negative gastric lavage culture, pneumothorax, impairing interpretation of form and cavity, with no symptoms.

Case 4, 7.9.48/1Ph456/R: Formation of exudate, negative gastric lavage culture with moderate symptoms. Previous digit 1 of table No. III indicating localization is altered to digit 4 in the same table, because of the pleural effusion involved.

SUMMARY

a) A classification of pulmonary tuberculosis with five digit code, is proposed for universal use.

b) Digit No. 1 concerns Tubercle Bacilli.
Digit No. 2 type and forms of the disease.
Digit No. 3 localization of lesions.
Digit No. 4 cavities and effusions, in pleura.
Digit No. 5 clinical status with symptomatology.

c) Comparison of five digit codes of the individual (same) examined at different times readily reveals worsening or improvement of condition, if the latest code contains digits that are higher than lower than before.

d) This five digit code classification is relatively simple and easy to use.

RESUMEN

a) Se propone una clasificación de la tuberculosis con un código de cinco índices para uso universal.

b) El Indice 1 se refiere al bacilo de la tuberculosis.
El Indice 2 al tipo y forma de la enfermedad.
El Indice 3 a la localización de las lesiones.
El Indice 4 a las cavidades y los derrames pleurales.
El Indice 5 al estado clínico y la sintomatología.

c) La comparación de los cinco índices en el mismo individuo examinado en tiempos diferentes, facilmente revela el empeoramiento o la mejoría si el código reciente proporciona o nó índices más o menos bajos.

d) La clasificación mediante el código o clave de cinco índices es relativamente sencilla y fácil de usar.

RESUME

1) L'auteur propose une classification de la tuberculose pulmonaire avec 5 index, pour emploi universel:

2) Index 1: concerne le bacille tuberculeux.
Index 2: type et formes de la maladie.
Index 3: localisations des lésions.

Index 4: cavités et participations pleurales.

Index 5: étude clinique et symptomatologie.

3) La comparaison de ces cinq index pour un même individu examiné en des temps différents révèle la régression ou l'amélioration de son état, selon que les indices du dernier code sont plus élevés ou plus bas qu'auparavant.

4) Cette classification en cinq indices est relativement simple et d'un emploi facile.

REFERENCE

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Standardized Oblique Roentgenography of the Chest

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In the roentgenographic examination of the chest, the oblique position is used infrequently because it has been used poorly. The lack of any standard of oblique angulation has resulted in haphazard placement which denies the two primary essentials of any chest film: (1) the ability to duplicate precisely for sequential study; (2) the presentation of a familiar pattern of anatomic topography.

The routine postero-anterior projection of the chest not only is standardized, but also nearly always will give some evidence of any existent intrathoracic abnormality. Full knowledge of the shape, size, position, and relationships of a lesion cannot be gained from any single projection. The supplementary film used most commonly is the lateral not only for the 90° comparison but also because its position can be duplicated. The lateral projection undoubtedly is helpful when the abnormality is known to be unilateral, well circumscribed, and dense. In many cases the combination of high penetration, bucky diaphragm, and superimposition makes the lateral either worthless or dangerously misleading.

When, in the fluoroscopic examination of a pulmonary lesion, the patient is placed so the abnormality is seen to best advantage, the position so assumed is rarely the postero-anterior or lateral, but usually one of intermediate obliquity. This same phenomonon will pertain to the positions used for the roentgenogram.

Properly positioned oblique films can "open" the obscure areas of hilum, apex, and left base with little sacrifice of detail. Collectively, the several films can present sufficient information and parallax not only to enhance the examination of the normal and questionable lung fields, but also for orientation of a lesion in its size, shape, and location.

These advantages can be gained and the difficulties of standardization can be obviated by the use of a simple apparatus.

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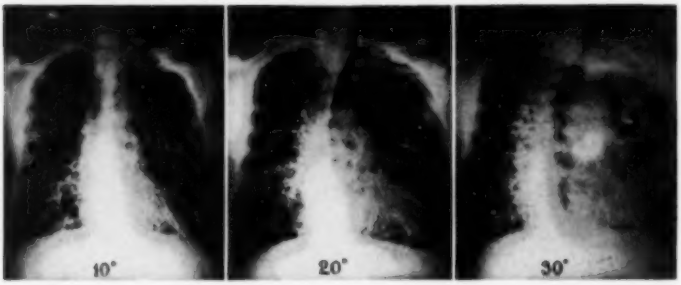

FIGURE 1: *Bronchograms Taken at Graded Angulations* show the most helpful projection to be intermediate. The 10° film is insufficiently turned while the 30° film has superimposition of the lung fields. *Comment:* The ideal position for a routine oblique film will be with the patient turned just to the superimposition point. The angle will be influenced geometrically by the depth of chest. Extremely deep chests may be shown best at 15°, while the unusually thin thorax will be best at 30°. The majority of patients can be taken satisfactorily at 25°. The angle used is indicated numerically on the marker for a permanent record. Note the unobstructed view of the right posterior apex.

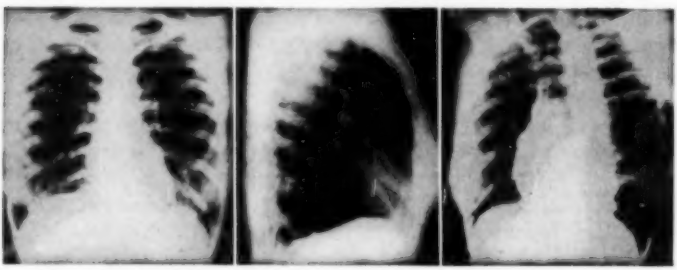

FIGURE 2: *The Lesion at the Left Base* was not thought to be hard and nodular until the oblique film was taken. Identification of the true nature of this abnormality led to removal of an early cancer.

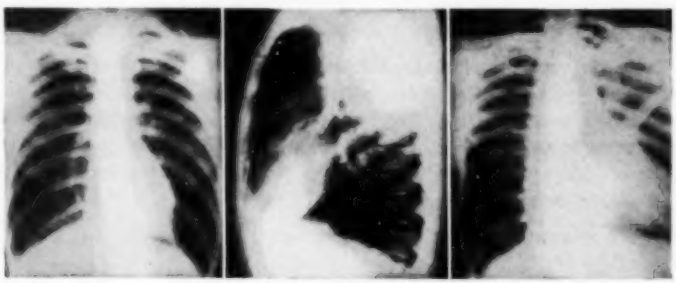

FIGURE 3: *The Cavity at the Right Apex* was never demonstrated until this 25° left posterior oblique film was taken.

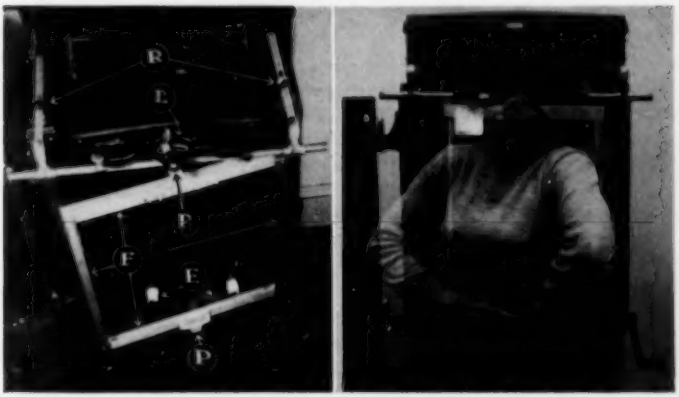

FIGURE 4: *Bird's Eye View* of the apparatus in place (left). A cassette-holding frame (F) is constructed to accept the type of cassette to be used. The top and bottom pieces are standard and can be obtained from any dealer in x-ray equipment. The frame is completed by adding sides of proper length. To provide angulation, the frame must have pivots (P) placed at the outside centers of the top and bottom pieces. These pivots must engage structure designed to maintain the frame so that its vertical axis (P-P) will remain approximately six inches from the face of the plate changer. The bottom pivot will engage a simple extension (E) which rests against the lower margin of the face of the plate changer. The top pivot will engage structure designed to hold the frame rigidly in place and also for easy attachment and detachment. This can be accomplished by various ways to fit different plate changers, many of which have sturdy top projections, such as shields, which would permit the simple hanging of a top extension similar to E. Lacking such a projection on the changer shown here, the problem was solved by rods-through-eyes supports (R), the eyes being permanently fastened to the top of the changer. Bands placed and tightened on the rods at the desired place limit the insertion of the rods by contact with the eyes. The rods are joined by a cross piece, which provides support for the upper pivot (P). The support and frame are coordinated through upper P by means of a protractor-pointer-lock device (L). The one shown here was salvaged from an old x-ray tube stand.

Tube's Eye View (right) of the apparatus with phantom exposure of patient whose back is aligned with cassette locked at 25° for a right posterior oblique film. The posterior position is always used for more precise placement and because the apex is well cleared of clavicular shadow when the tube is centered at the level of the xiphoid.

SUMMARY

By use of a simple apparatus, the oblique projection can be standardized and thus enhance complete roentgenographic examination of the chest. Ideally, similar apparatus could be applied to the fluoroscopic arm and screen, so that any desirable view in fluorescence could be duplicated in the roentgenogram. The proper and complete use of standard projections can provide valued information, and often make unnecessary the resort to complex and costly forms of examination.

RESUMEN

Por el uso de un aparato simple la proyección oblicua puede estandarizarse y así obtenerse un examen completo del tórax.

Idealmente un aparato similar podría aplicarse al brazo fluiroscópico y a la pantalla de modo que cualquier aspecto a la fluoroscopía podría duplicarse en la película.

El uso de formas estandarizadas de proyección puede proporcionar valiosa información que a menudo haría innecesarias otras formas de examen más complicadas y costosas.

RESUME

La projection oblique peut être standardisée par l'utilisation d'un dispositif simple. Ainsi se trouve renforcée la valeur de l'examen radiographique du thorax. L'idéal serait qu'un dispositif semblable puisse être adapté à l'écran, de façon à ce que tous les aspects qui semblent intéressants à la radioscopie puissent être reproduits sur un cliché.

L'utilisation convenable et complète des clichés standard peut donner des renseignements de valeur, et rend souvent inutile le recours à des modalités d'examen plus compliquées et plus onéreuses.

Benign Fibroma of Mediastinum

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There is no absolute criterion as to differentiation between a malignant and benign tumor of the thorax, but all such lesions should be evaluated for surgical resection. Even though the tumor may be benign, it may eventually lead to serious complications due to pressure on other vital organs, infection, or undergo malignant degeneration. In 1941 Blades reported 32 cases of intrathoracic fibromas from the world's literature.

Tumors of the mediastinum are generally classified according to their location. Bradford et al. classifies tumors into congenial and acquired cysts, connective tissue tumors, neurogenic tumors, thymic tumors, primary tumors of lymph nodes, and intra-thoracic goitres. Most tumors in the posterior mediastinum are neurogenic in origin, while those in the anterior mediastinum are teratomas or lymphomas. Other tumors which may occur are bronchial cysts, pericardial cysts, connective tissue tumors, thymomas, and mediastinal goitres.

Differential diagnosis of primary mediastinal tumors is usually difficult, and there is no characteristic symptom complex. In the early stages all are asymptomatic, and in the benign group the symptoms are produced by enlargement resulting in pressure. Benign primary tumors of the mediastinum are usually found on routine x-ray inspection of the chest. While x-ray studies are most important in locating them, other procedures such as bronchoscopy, diagnostic pneumothorax, and angiocardiography may be helpful. All intrathoracic undiagnosed lesions should have bronchoscopy prior to exploratory operations.

Report of Case

A 40 year old white female (48234) was admitted to the Columbia Hospital on April 25, 1950, with no specific complaint referable to the chest. She had consulted her physician because of an acute respiratory infection and x-ray inspection of the chest revealed a tumor mass on the right side. The respiratory infection subsided on chemotherapy and she was referred for treatment of the chest tumor. The past history and family history were not significant.

Physical examination revealed a well developed, slender, undernourished white female, not apparently ill. The temperature, pulse and respiration were normal. No pertinent abnormal finding was elicited on complete physical examination.

Laboratory studies revealed a normal blood count with 85 per cent hemoglobin. Urinalysis and blood Wasserman were negative.

Roentgenographic studies showed evidence of a small rounded soft tissue mass in the posterior mediastinum just to the right of the dorsal vertebra. The mass measured 6 centimeters in greatest diameter and extended from the sixth to the eighth ribs posteriorly. Barium swallow showed that this mass had no relationship to the esophagus. Flouroscopy revealed no visible pulsation of the mass. Other chest structures were negative. There was no evidence of bone erosion or destruction in the dorsal vertebra or ribs.

Thoracotomy was performed under endotracheal ether-oxygen anesthesia with pentothal induction. The posterior half of the seventh rib was resected subperiosteally. The underlying parietal pleura was opened throughout the extent of the incision. The lung collapsed normally, and appeared normal on palpation and inspection. There were no adhesions or inflammatory reaction. Lying subpleurally in the posterior mediastinum there was a large smooth tumor mass at the level of the sixth to

FIGURE 1

FIGURE 2

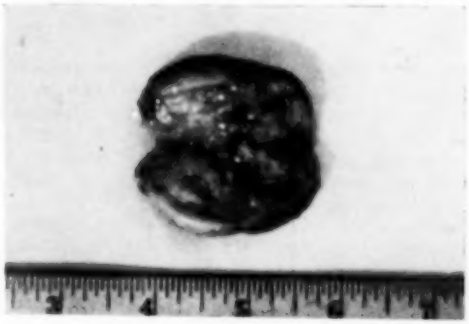

FIGURE 3

the eight ribs. The mediastinal pleura was opened, and a line of cleavage was developed between the tumor mass and the parietal mediastinal pleura. The pleura was somewhat adherent to the tumor and bled freely on seperation. The tumor was completely excised intact. No pedicle could be identified.

She made an uneventful postoperative recovery, and was discharged from the hospital on May 3, 1950. One month later she was in good condition, and has now resumed her occupation.

Pathological Report

Received a well encapsulated, brownish to red colored, biscuit shaped tumor mass, measuring 5 cms. in diameter and 2½ cms. in thickness. Gross sections through the tumor revealed considerable resistance to a very sharp knife, and the cut surfaces were white, to gray, to brownish in color, with an irregular, mottled pattern throughout. There were a few ragged open spaces in the tissue and there was at least one area of hemorrhage which may have resulted during surgical removal of the tumor.

Microscopic sections show a well encapsulated, fibrous connective tissue growth which is undergoing very extensive liquefaction necrosis. In the few areas where necrosis is not present the tumor cells are long, slender, spindle or cigar shaped and tend to grow in parallel bundles of considerable size. The nuclei of these cells are long, and slender and spindle shaped, and take a very deep stain. No mitotic figures are noted. In the areas of necrosis there is a rough, pale blue staining collaganeous background in which are many open spaces or vesicles, which present great irregularity of size and outline. The blood vessels of the tissue are not numerous, but are well developed, and the smaller ones present a moderate degree of intimal thickening. In a few portions are seen a few infiltrating lymphocytes. There is one rather large area of hemorrhage in the tissue. There is no evidence of malignancy in the sections. *Diagnosis:* Fibroma of mediastinum, undergoing liquefaction necrosis.

Discussion

Conclusive evidence as to exact origin of the tumor reported in this case is lacking. It is reported as a mediastinal tumor because it was medial to the mediastinal pleura, although it was adherent to the parietal pleura by fibrous bands, but without a definite pedicle. This tumor could arise from the sub-serous tissue of the parietal mediastinal pleura and thus be classified as a solitary benign tumor of the pleura. It could also have had its origin from the sheaths of the sympathetic nerves or ganglia and be classified as a neurilemoma. Anatomically, it is similar to four other cases to be reported in another study of solitary pleural tumors.

SUMMARY

1) A case of benign connective tissue tumors occurring in the mediastinum is reported.

2) There is no conclusive evidence as to the site of origin, and it may be a benign solitary tumor of the parietal pleura, or a neurilemoma derived from sheaths of sympathetic nerves.

3) It is believed that all tumors of the mediastinum should be evaluated for resection, because those that are benign may continue to grow and interfer with vital organs in the thorax.

4) This case demonstrates the value of routine photofluoroscopy and x-ray film studies of the chest in the detection of early lesions.

RESUMEN

1) Se informa sobre un caso de tumor benigno de tejido conectivo que ocurrió en el mediastino.

2) No existen pruebas concluyentes respecto al sitio de origen, y es posible que haya sido un tumor solitario benigno de la pleura parietal o un neurilemoma derivado de las vainas de los nervios simpáticos.

3) Se opina que se debe considerar la resección en todos los tumores del mediastino, pues los que son benignos pueden continuar creciendo hasta que causen daño a los órganos vitales del tórax.

4) Este caso demuestra el valor de la fotofluorografía y de los estudios roentgenográficos sistemáticos del pecho en el descubrimiento de lesiones tempranas.

RESUME

1) L'auteur rapporte un cas de tumeur bénigne du tissu conjonctif apparue dans le médiastin.

2) L'origine exacte de cette tumeur n'est pas démonstrée avec évidence et il peut s'agir d'une tumeur bénigne de la plévre pariétale ou d'un neurinome provenant des nerfs sympathiques.

3) L'auteur pense que toutes les tumeurs du médiastin doivent être l'objet d'une intervention chirurgicale. En effet, celles qui se présentent comme bénignes peuvent cependant continuer à augmenter de volume et amener des troubles dans les organes essentiels du thorax.

4) Ce cas démontre la valeur des examens radiophotographiques et radiographiques du thorax pratiqués à titre systématique pour découvrir précocemment certaines lésions.

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Migratory Pneumonia Without Eosinophilia*

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Migratory pulmonary infiltrations with eosinophilia is a well-known clinical entity. The patient is rarely if ever severely ill and there is always a peripheral eosinophilia. This case, however, differs from those usually classified as Loeffler's syndrome by reason of its rather severe clinical course, and the absence of eosinophilia.

Mrs. K. S., a 56-year old housewife entered Irvington General Hospital on May 19, 1949 with complaints of fever and a dry, hacking cough of about 10 days duration. Until this entry, she could not recall any previous febrile illness, nor indeed, any illness since childhood. There was no history of allergy. She could not remember when she had last consulted a physician, but believed not since at least 30 years.

Physical examination revealed a middle-aged woman acutely ill, but in no apparent distress. Her temperature was 101.8 degrees F. Many rales were heard in both lower lung fields. There were no other findings that were deemed important. The diagnosis was primary atypical pneumonia.

For the 37 days this patient remained in the hospital, only during the last three was she afebrile. The first 34 days she suffered malaise, weakness, and fatigue. Her appetite was poor and she had a dry cough.

After the first week marked bronchial breathing was heard in the right lower lobe. This finding persisted until about one week before she was discharged.

At the end of the third week cyanosis of the lips became evident, and persisted for a few days during which time she appeared to be critically ill.

Because sputum was scanty, specimens were obtained with difficulty. Gastric aspiration was refused. For a certain period potassium iodide was administered in an attempt to increase the amount of sputum, which was found to contain no acid-fast bacilli and no fungi.

On the day of admission penicillin was administered, and three days later streptomycin was begun as well. On the 16th day after the first administration of penicillin, and the concurrent course of streptomycin, both antibiotics were discontinued since they appeared to have had no beneficial effect. Twice a course of aureomycin was begun, and twice it was discontinued because the patient's low tolerance for this antibiotic caused nausea, and it appeared that she was receiving no benefit from the drug. Chloromycetin was administered, but that too was discontinued after a trial which showed no beneficial result.

Blood smears taken on the second hospital day showed 2 per cent eosinophiles, with a total white count of 12,400. On the 15th hospital day results of laboratory tests showed that the eosinophile count remained

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at 2 per cent, with 10,800 leukocytes. Several blood cultures were found to be sterile. Heterophile antibody reaction was negative on the 20th day.

On the 34th day she became afebrile, and her temperature did not rise again. Her malaise cleared, and her appetite improved so that within a few days she felt well enough to be discharged. About this time all physical signs in the chest cleared as well.

After discharge she remained afebrile. For a short time she suffered weakness, but soon regained strength, and though her general improvement was so gradual as to make convalescence protracted, when last seen about three months following discharge, she claimed to be feeling well.

The x-ray film of the lungs taken on admission showed a light diffuse infiltration of the right lower and middle lobes (Figure 1a). There was some dense consolidation in the left lower lung field. The x-ray film of

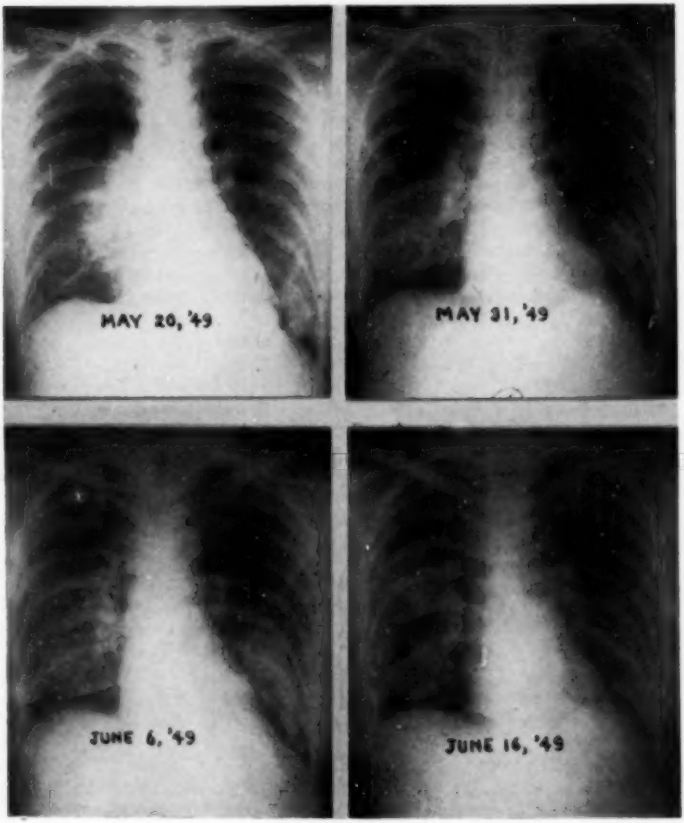

FIGURE 1

May 31 (Figure 1b) showed a shadow on the right to be a little lighter; that on the left seemed denser and slightly more extensive. The June 6th film (Figure 1c) shows the process on the right to be extending upward. Consolidation in the left base still appears dense, and has extended somewhat. The June 16th film (Figure 1d) shows the right upper lobe to be definitely involved, while the lesion in the lower part on the right appeared to be clearing. There seemed to be a slight diminution in density of the lesion on the left.

On July 2, 1949, eight days after her discharge further x-ray films were made. In Figure 2a it may be noted that the lesion in the right upper lobe has apparently cleared, but there is little change in the lower part of that lung. The consolidation in the left lung, however, has extended upward. Considerable clearing of the right lung is evident (Figure 2b) in the July 16th films. There now remains but a small infiltration at

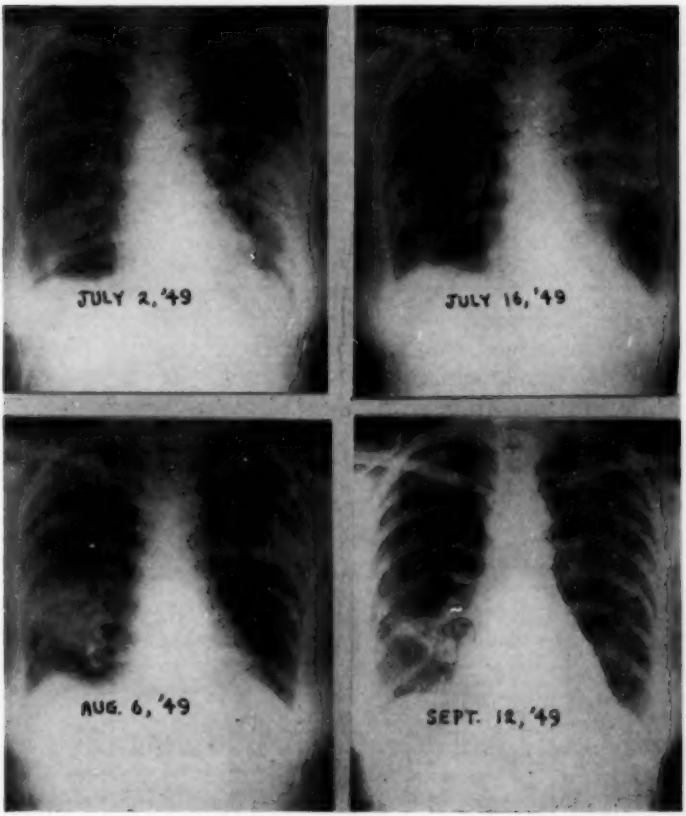

FIGURE 2

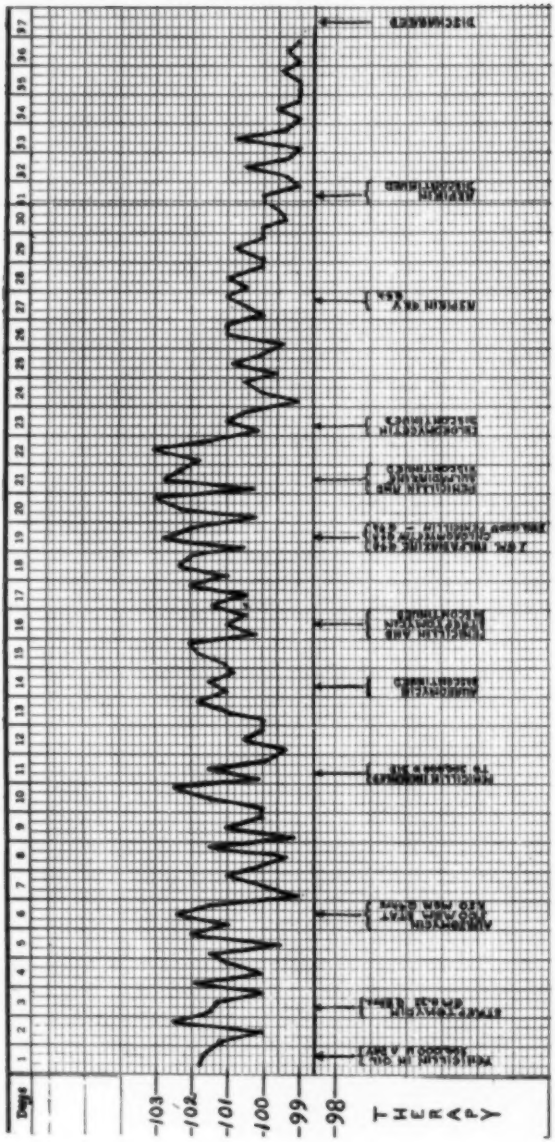

FIGURE 3

the base. The left lung, however, shows a clear base with widespread dense consolidation of the upper portion. On August 6th (Figure 2c) the base of the right lung still showed considerable consolidation, with tenting of the diaphragm at one point. The consolidation previously seen on the left had largely cleared.

This patient was last seen in September 1949. X-ray films taken on the day of her examination, September 12th, showed regression of all lesions that had been noted previously. A few scattered linear markings could be seen. Both diaphragms were smooth and no tenting was evident. Agglutination tests made on that day were negative for typhoid, paratyphoid, brucella OX_2 and OX_{19}. Complement fixation for "Q" fever was also negative.

The patient declared herself to be in excellent health, and claimed she felt as well as she had before the onset of her illness.

SUMMARY

1) An illness characterized by migratory pulmonary consolidation without eosinophilia is described.

2) Contrary to those cases usually classified as Loeffler's syndrome, in which the clinical course is not severe and there is always peripheral eosinophilia, this patient had a rather severe clinical course with a complete absence of eosinophilia.

3) Antibiotics were ineffective; the etiology remains unknown.

RESUMEN

1) Se describe un mal caracterizado por una consolidación pulmonar migratoria sin eosinofilia.

2) Contrariamente a aquellos casos que generalmente se clasifican como síndrome de Loeffler, en los que el curso clínico no es grave y en los que hay siempre una eosinofilia periferal, este paciente tuvo un curso clínico bastante grave con una ausencia completa de eosinofilia.

3) Los antibióticos fueron ineficaces y la etiología queda desconocida.

RESUME

1) Description d'une affection caractérisée par des infiltrats pulmonaires labiles sans éosinophilie.

2) Ces malades ont été atteints d'une affection assez sévère avec absence complète d'éosinophilie. Ces faits sont en opposition avec les observations habituellement considérées comme appartenant au syndrome de Loeffler; dans ce cas en effet, l'évolution est en général bénigne, et il existe toujours une éosinophilie sanguine.

3) Les antibiotiques furent sans action. L'étiologie demeure inconnue.

Spontaneous Rupture of the Esophagus*

HENRY L. RIGDON, M.D. and N. DOUGLAS ELLIS, M.D.
Florence, South Carolina

Perforation of the esophagus for any reason or at any level is always a grave lesion and one which, if untreated, is not compatible to life. The clinical symptoms of spontaneous rupture of the esophagus are not generally recognized. The majority of the cases thus far recorded in the literature, now more than 66, were not diagnosed before death and in many instances were not even suspected before the postmortem examination.

Since the first classical description of the condition by Boerhave[1] published in 1724, there have appeared in the literature isolated cases of this condition. Eliason and Welty[2] reported three fatal cases of spontaneous rupture of the esophagus and reviewed the theories as to their etiology, plus the usual clinical and autopsy findings. Apparently the predominating cause is the sudden increase in intra-esophageal pressure brought on by forceful vomiting, straining or external trauma to the abdomen or thorax. In practically all cases coming to autopsy a linear tear is found in the esophagus just proximal to the cardia. Associated with this are variable degrees of mediastinitis and pleuritis. Whether or not preexisting pathologic changes must be present in the esophagus wall before rupture can occur has not been decided definitely.

More recent reports include that of Scholefield[3] who described two cases that were diagnosed correctly. One patient died before surgery could be performed; the other was operated upon and recovery was prompt. This was the third successful surgical repair recorded in the literature.

Cliffton[4] recently reported three cases, one of which was treated successfully by surgery; the second, a fatal case of rupture, was diagnosed incorrectly; the third case was diagnosed as a spontaneous perforation of the esophagus and was treated by laparotomy, with survival after late diagnosis and thoracotomy drainage.

It is generally accepted that thoracotomy with primary repair of the rupture is the procedure of choice, especially in tears in the intrathoracic portion of the esophagus. However, Temple[5] advocates expectant therapy in cervical perforations, especially where the tear is small and easily accessible.

*From the Department of Surgery, The McLeod Infirmary, Florence, South Carolina.

Frink,[6] in recording a case of spontaneous rupture of the esophagus with recovery following thoracotomy drainage, reviewed the literature and pointed out that most frequently the patient wih a ruptured esophagus is a male between 35 and 40 years of age who has been a heavy drinker. After an episode of forceful vomiting with or without hematemesis the patient is seized with a sudden agonizing upper abdominal substernal pain so severe that the usual dose of morphine gives no relief. An important observation is the early presence of subcutaneous emphysema in the neck and face. At the time of his report Frink wrote that there has been no report of survival in a patient with spontaneous rupture.

Moore and Murphy[7] reported one patient with spontaneous rupture who recovered following laparotomy and thoracotomy drainage, and later primary repair of the esophageal tear.

Case Report

A 38 year old white male was admitted to the hospital on February 9, 1950 with the complaint of mild epigastric pain and a history of having vomited a small amount of bright red blood a few hours previously. He volunteered the information that he had been drinking rather heavily for a few days and had not eaten adequately. He said that five years previously he had a roentgenographic examination at another hospital. A diagnosis of peptic ulcer was made and he was placed on a diet, but since then there were no symptoms suggestive of ulcer. The past history and family history were noncontributory.

A physical examination revealed a well nourished white male of 38 in no apparent distress. His blood pressure was 122 mm. of mercury systolic and 74 mm. diastolic. No abnormal physical findings were noted other than slight epigastric tenderness. Urinalysis was essentially negative and the blood count was within normal limits.

He was admitted to the hospital for overnight observation and placed on a bland diet. Approximately six hours after admission he complained of dyspnea, cyanosis, and severe chest pain. Examination at this time showed that there was definite emphysema of the neck and he was drenched in sweat. He was cyanotic and it was apparent that he was suffering excruciating pain. This pain had started a few minutes previously when he vomited a small amount of bright blood. The chest was resonant throughout, although there was a decrease in breath sounds which was attributed to shallow respiration. A tentative diagnosis of spontaneous pneumothorax with mediastinal emphysema or a ruptured esophagus was made.

The patient was given one-half grain of morphine. A roentgenographic examination was made with the following findings: "Barium was given and passed down the esophagus, then appeared to leave the esophageal lumen above the diaphragm. The films show marked emphysema throughout the mediastinum and neck. The barium appears to extravasate from the lower esophagus and is shown filling the lower mediastinal tissues above the diaphragm to the size of a tangerine. Diagnosis: probably ruptured esophagus."

A fluoroscopic examination was made approximately two hours after the onset of pain. Plans were made for immediate operation, he was matched for blood, and oxygen was administered by intranasal catheter. Under intratracheal cyclopropane-oxygen-ether anesthesia the left pleural cavity was entered through the eighth interspace. The mediastinal tissues were found to be edematous and discolored from barium, gastric juices, and bile. The mediastinal surface of the lung was likewise discolored, although apparently the process had not ruptured through into the pleural cavity. The mediastinal pleura was divided and the esophagus exposed directly above the diaphragm. There was a laceration about two and one-half cm. long just over the diaphragm on the left side of the esophagus. A finger was placed in this rent and was found to enter into the cardia of the stomach. This perforation was closed with two layers of interrupted cotton sutures. The pleural cavity was then drained by inserting a No. 18 rubber catheter in the ninth interspace and bringing it out through a stab wound. The chest was closed in layers and all excess air aspirated from the left pleural cavity.

Immediately after operation the patient appeared cyanotic. His pulse was rapid and almost imperceptible. It became apparent that there was some obstruction to the right major bronchus. The left lung was partially collapsed as a result of the pneumothorax caused by the operation. The patient expired before bronchoscopy could be performed.

Postmortem Findings

A postmortem examination showed that extending upward 9 cm. from the cardiac opening there was a soft striated light grayish red to grayish white and yellow tissue. At a distance of 2.5 cm. above the cardia a small opening was found in the tissue about the esophagus, from which a thin bloody fluid exuded. Four cotton sutures were visible along the left border of the esophagus.

Considerable emphysema and bubbly aeration of the fat were seen anterior to the pericardium and about the root of both lungs. Some of these lobules of fat were distended to form prominent lugs.

Examination of the right side of the esophagus, the posterior surface of the root of the right lung, and the tissue upward from the diaphragm revealed no evidence of trauma.

The right lung was dark purplish red and doughy, except for blotchy light grayish red areas over the upper and middle lobes.

There was a small area of pinkish gray crepitant lung at the apex of the upper lobe of the left lung, otherwise the tissues of both lungs appeared to have entirely collapsed. Crepitation was present throughout all of the tissues of the mediastinum.

Dark red submucosal hemorrhage extended 5 cm. above the upper end of the repaired area. The mucosa was bluish gray and finely granular, and revealed superficial denudations. Above this level the mucosa and wall were normal.

An incision into the right bronchus and trachea showed the tubes to be filled with a dark red jelly-like blood clot mixed with gray mucus. This material also entirely filled the left bronchus. The trachea was opened to the larynx and contained the same type of material.

Dissection of the neck was made and the pharynx opened. Thin blood was found draining down the posterior pharyngeal wall from the nasopharynx.

The tissue from the right lung floated in water; that from the left lung sank in water.

The air passages were obstructed completely by blood and mucus, as previously mentioned.

There was no evidence of injury to the epiglottis, larynx, trachea or bronchi to account for the large amount of clotted blood found.

The extensive light pinkish gray to grayish yellow softening seen in the tissues about the left bronchus and the lower esophagus was caused by the digestive action of gastric juice.

There was no evidence of erosion, ulceration or thickening of any part of the esophageal wall.

No interesting observations were made other than the hemorrhages noted in the wall of the esophagus above the repaired tear.

Comment

Surgical repair of the esophagus following spontaneous rupture is the radical procedure to follow on a desperately ill patient. To be successful it must be done within the first few hours after the rupture has taken place, when an accurate diagnosis usually is most difficult.

It is felt that the patient reported here would have had an excellent chance of recovery if the trachea and bronchus had not become occluded by blood; the origin of this blood in the bronchi is as yet unexplained. Even after autopsy it was impossible to determine its origin.

In retrospect it was noted at the time of operation that there was a blood stain on the right side of the patient's face near he mouth. It was assumed that this was the result of the vomiting of blood. No areas of trauma were observed in the nasopharynx or trachea that could possibly have bled as a result of trauma from introduction of the intratracheal tube. It is extremely regrettable that this patient died from such an unusual cause after an early diagnosis and a surgical repair that was carried out within three hours of perforation.

SUMMARY

1) A case of spontaneous rupture of the esophagus is reported.

2) It must be emphasized that although this is a rare condition and one that produces a high rate of mortality, it should be considered in differential diagnosis in patients with severe upper abdominal pain or pain in the chest associated with emphysema of the neck and face.

RESUMEN

1) Un caso de ruptura espontánea del esófago se refiere.

2) Debe recalcarse que aunque es un accidente raro y que produce una elevada mortalidad, debe ser considerado su diagnóstico

diferencial en enfermos con dolor intenso en el abdomen alto o en el tórax asociado con enfisema de la cara y del cuello.

RESUME

1) Les auteurs rapportent une observation de rupture spontanée de l'oesophage.

2) Il s'agit d'un accident rare dont l'évolution est mortelle dans une grande proportion. Cependant il y a lieu de discutar ce diagnostic chez les malades qui sont atteints de douleur violente de la partie supérieure de l'abdomen ou de douleur thoracique associée à un emphysème du cou et de la face.

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Editorial

PSYCHOSOMATIC MEDICINE

Dr. Arnold S. Anderson presents a very interesting and unique article in this issue. In his treatment of psychogenic factors in chest disease he emphasizes two important points. First, that there is abundant evidence of psychogenic factors in chest disease, and second, the human attributes with which a physician should be endowed if he is to successfully deal with the many psychogenic problems of the chronic invalid.

I am sure that most any psychiatrist and internist will agree with Dunbar's statement "we know now that bodily changes may be brought about by mental stimuli, by emotion, just as effectively as by bacteria and toxins, and that psychological changes accompanying emotion may disturb the function of any organ in the body."

Since most acute infectious diseases are now practically controlled by the use of vaccines, antibiotics, and public health measures, together with increased longevity, we are of necessity more concerned with chronic and degenerative diseases. We are forced to recognize the urgent need to study and deal with the ever increasing number of patients whose complaints and invalidism are brought about by emotional distress. It is also important to understand that one etiological factor is the imbalance of the internal environment as well as the physical environment.

In chronic diseases of the chest, especially in pulmonary tuberculosis, more and more studies are being made from the psychogenic angle. It is believed that anxiety with all its implications is the one factor present in every patient with chronic chest disease. In the case of pulmonary tuberculosis it has recently been shown that anxiety in varying degrees is also found in all arrested cases of tuberculosis. Therefore, psychosomatic approaches must be made in the rehabilitation of these patients.

From observations that have been made by internists and by foremost psychiatrists it is becoming more generally recognized by the medical profession that mental therapy is nearly as essential as physical and medical treatment in the curing of pulmonary tuberculosis.

Untrained in dynamic psycho-pathology and the techniques of investigating and treating problems and interpersonal relationships, the internist has found an ally in the psychiatrist. Psychiatry at the same time has stopped being preoccupied with a

search for physiological causes for emotional imbalance and has become intensely interested in the psychological concomitants of emotional maladjustment. It was the common scientific pursuit, as well as the old interest in the patient as a person, which led to coordination of interests.

Dr. Anderson describes his ideal physician who would become an ideal psychosomatist. All of the traits he mentions are valuable, and I would add that the physician of the future be trained in the psychosomatic approach.

The development of psychosomatic medicine and the organization of programs in comprehensive medicine in the liason relationships between the department of psychiatry and the department of medicine have created problems concerning instruction in the use of the psychosomatic approach.

There are two groups who should receive such instruction, the physician already in practice and the medical students. There are now institute courses in psychosomatic medicine available for the general practitioner, Armed Forces medical officers, and Public Health officers at the University of Minnesota, University of Cincinnati, Fitzsimmons, Letterman, Walter Reed General Hospital, and the California Institute on Mental and Public Health. The Duke University School of Medicine has made some studies concerning the methods to be used in teaching psychosomatic medicine to house officers.

It would seem that the ideal instructor for the medical student would be a highly trained internist who later has become a psychiatrist in the final stages of his medical training. The problem of organizing methods of instruction in this all important subject is still in the trial and error stage. In due time, however, we can be sure a complete renovation of the curriculi in our medical schools will bring about a more comprehensive integration of psychiatry with other departments in medicine.

C. M. Hendricks, M.D., F.C.C.P.

SEVENTEENTH ANNUAL MEETING

American College of Chest Physicians

The Seventeenth Annual Meeting of the College will open on Thursday, June 7 at the Ambassador Hotel, Atlantic City, New Jersey. During the opening day administrative meetings and examinations for Fellowship in the College will take place.

The scientific sessions of the meeting will begin on Thursday evening, June 7, at 8:00 p. m. Due to the amount of interesting material submitted this year, the scientific program committee has scheduled the opening session for Thursday evening instead of Friday morning, which has been the custom at previous meetings. The scientific sessions will continue through Friday, Saturday and Sunday, June 8, 9 and 10. An outline of the complete program follows:

Wednesday, June Sixth

6:00 p.m.—*Registration*
The Registration Desk will be open from 6:00 to 8:00 p.m. for the convenience of members arriving in Atlantic City on Wednesday.

8:00 p.m.—*Executive Council Meeting*
Louis Mark, Columbus, Ohio, President, presiding.

Committee on Board Certification
J. Winthrop Peabody, Washington, D. C., Chairman.

Committee on College Award
Andrew L. Banyai, Milwaukee, Wisconsin, Chairman.

Board of Examiners
Alvis E. Greer, Houston, Texas, Chairman.

Thursday, June Seventh

8:00 a.m.—*Registration*
Please register early. There is no registration fee. It is essential that you obtain your tickets for the luncheons and the President's Banquet at the time you register as the supply of tickets is limited.

ADMINISTRATIVE SESSIONS:

10:30 a.m.—*Board of Governors Meeting*
William R. Rumel, Salt Lake City, Utah, Chairman, presiding.

2:00 p.m.—*Board of Regents Meeting*
James H. Stygall, Indianapolis, Indiana, Chairman, presiding.

COUNCIL AND COMMITTEE MEETINGS

8:00 a.m.—*Board of Examiners*
Alvis E. Greer, Houston, Texas, Chairman, presiding.

9:00 a.m.—*Council on Postgraduate Medical Education*
J. Winthrop Peabody, Washington, D. C., Chairman, presiding.

9:00 a.m.—*Council on Undergraduate Medical Education*
Edward W. Hayes, Monrovia, California, Chairman, presiding.

2:00 p.m.—*Council on Management and Treatment of Diseases of the Chest*
Edwin R. Levine, Chicago, Illinois, Chairman, presiding.

Committee on Surgical Treatment of Diseases of the Chest
Alfred Goldman, Los Angeles, California, Chairman.

THURSDAY, JUNE SEVENTH (Continued)

Committee on Non-Surgical Collapse Therapy
Harold G. Trimble, Oakland, California, Chairman.

Committee on Chemotherapy and Antibiotics
Karl H. Pfuetze, Chicago, Illinois, Chairman.

Committee on Physiologic Treatment
Alvan L. Barach, New York City, Chairman.

2:30 p.m.—*Council of Tuberculosis Hospitals*
Russell S. Anderson, Erie, Pennsylvania, Chairman, presiding.

Committee on Sanatorium Standards
I. D. Bobrowitz, Otisville, New York, Chairman.

Committee on Rehabilitation
Allan Hurst, Denver, Colorado, Chairman.

Committee on Hospital Statistics
William L. Potts, Columbus, Ohio, Chairman.

4:00 p.m.—*Council of Tuberculosis Committees*
James H. Stygall, Indianapolis, Indiana, Chairman, presiding.

4:00 p.m.—*Council on Public Relations*
Edgar Wayburn, San Francisco, California, Chairman, presiding.

Committee on College Essay
Eli H. Rubin, New York, New York, Chairman.

4:00 p.m.—*Committee on Membership*
Chevalier L. Jackson, Philadelphia, Pa., Chairman, presiding.

5:00 p.m.—*Council on Public Health*
Robert J. Anderson, Washington, D. C., Chairman, presiding.

Committee on Chest Diseases in Institutions
Otto L. Bettag, Chicago, Illinois, Chairman.

5:00 p.m.—*Committee on College By-Laws*
Charles M. Hendricks, El Paso, Texas, Chairman, presiding.

5:00 p.m.—*Council on International Affairs*
Chevalier L. Jackson, Philadelphia, Pa., Chairman, presiding.

Council on Pan American Affairs
Richard H. Overholt, Brookline, Massachusetts, Chairman.

Council on European Affairs
Andrew L. Banyai, Milwaukee, Wisconsin, Chairman.

Council on Pan Pacific Affairs
Seymour M. Farber, San Francisco, California, Chairman.

Council on African and Eastern Affairs
Edgar Mayer, New York, New York, Chairman.

6:00 p.m.—*Editorial Board*
Jay Arthur Myers, Minneapolis, Minnesota, Chairman, presiding.

EXAMINATIONS FOR FELLOWSHIP

9:00 a.m.—*Oral Examinations*

2:00 p.m.—*Written Examinations*

The Board of Examiners will conduct the oral and written examinations for Fellowship in the American College of Chest Physicians. Candidates are requested to report at 8:30 a.m. at the Examining Room as designated on the hotel bulletin board.

12:00 Noon—LUNCHEON MEETING

THURSDAY, JUNE SEVENTH (Continued)

SEVENTH ANNUAL CONFERENCE OF COLLEGE CHAPTER OFFICIALS, BOARD OF REGENTS AND BOARD OF GOVERNORS
Martin H. Collier, Blackwood, New Jersey, presiding.

Awarding of Certificates of Merit to Past-Presidents of College Chapters
Louis Mark, Columbus, Ohio, President.

Report, Conference of College Chapter Officials
Andrew L. Banyai, Milwaukee, Wisconsin, Chairman,
George R. Hodell, Houston, Texas, Secretary.

Friday, June Eighth

5:00 p.m.—*Meeting, Committee on Nominations*
Jay Arthur Myers, Minneapolis, Minnesota, Chairman, presiding.

6:00 p.m.—*Meeting, Council on Research*
Charles M. Hendricks, El Paso, Texas, General Chairman, presiding.

Council on Research, Scientific Section
Alvan L. Barach, New York, New York, Chairman.

Council on Research, Financial Section
Jay Arthur Myers, Minneapolis, Minnesota, Chairman.

The Board of Directors of the American Research and Education Foundation for Chest Disease will meet with the Council on Research of the College.

Saturday, June Ninth

9:00 a.m.—*Administrative Session*
Louis Mark, Columbus, Ohio, President, presiding.

Report of the Treasurer
Benjamin L. Brock, Downey, Illinois.

Report of the Committee on Nominations
Jay Arthur Myers, Minneapolis, Minnesota, Chairman.

Report of the Committee on College By-Laws
Charles M. Hendricks, El Paso, Texas, Chairman.

Report of the Executive Secretary
Murray Kornfeld, Chicago, Illinois.

The Administrative session is open to all members of the College.

Sunday, June Tenth

9:00 a.m.—*Meeting, Joint Committee on Industrial Diseases of the Chest*

American Association of Industrial Physicians and Surgeons
Arthur J. Vorwald, Saranac Lake, New York, Chairman.

American College of Chest Physicians
Louis L. Friedman, Birmingham, Alabama, Chairman.

5:00 p.m.—*Meeting, Board of Regents*
James H. Stygall, Indianapolis, Indiana, Chairman, presiding.

Monday, June Eleventh

6:30 p.m.—*Dinner Meeting*
Joint Committee on Chest X-Ray

American College of Radiology
Leo G. Rigler, Minneapolis, Minnesota, Chairman.

American College of Chest Physicians
Otto L. Bettag, Chicago, Illinois, Chairman.

Scientific Program

Thursday Evening, June Seventh

8:00 p.m.—*Scientific Session No. 1*
Irving Willner, Newark, New Jersey, Chairman,
John S. Packard, Allenwood, Pennsylvania, Secretary.

"Broncholithiasis,"
Coleman B. Rabin and Morton Ziskind, New York, New York.
Discussor: Chevalier L. Jackson, Philadelphia, Pennsylvania.

"The Use of Crystalline Trypsin in Chest Disease,"
L. Chandler Roettig, Howard Reiser and
William Habeeb, Columbus, Ohio.
Discussor: Louis Mark, Columbus, Ohio.

"The Management of Certain Problems in Pulmonary Resection,"
Donato G. Alarcon, Mexico City, Mexico.
Discussor: J. Maxwell Chamberlain, New York, New York.

"Newer Palliative Procedures in the Treatment of Inoperable Thoracic Tumors,"
Edgar Mayer and Bernard Roswit, New York, New York.
Discussor: A. Lee Lichtman, New York, New York.

Friday, June Eighth

9:00 a.m.—*Scientific Session No. 2*
William E. Ogden, Toronto, Canada, Chairman,
Darrell H. Trumpe, Springfield, Illinois, Secretary.

"Primary Tuberculosis as a Cause of Bronchiectasis in Children,"
Lloyd B. Dickey, San Francisco, California.
Discussor: W. L. Howard, Northville, Michigan.

"A Study of Delayed Non-Specific Tuberculin Reactions,"
M. R. Lichtenstein and Carmine Cilella, Chicago, Illinois.
Discussor: Edward A. Piszczek, Chicago, Illinois.

"The Effect of the Diminished Incidence of Primary Infection on the Tuberculosis Control Program,"
Jay Arthur Myers, Minneapolis, Minnesota.
Discussor: Robert J. Anderson, Washington, D. C.

"Studies of Respiratory Air Flow in the Measurement of Ventilatory Function,"
Donald F. Proctor, Baltimore, Maryland.
Discussor: George R. Meneely, Nashville. Tennessee.

"Physiologic Factors in the Use of the Body Respirator for Impaired Respiratory Function,"
James L. Whittenberger and James V. Maloney, Jr.,
Boston, Massachusetts.
Discussor: Alvan L. Barach, New York, New York.

"Pectus Excavatum,"
Francis M. Woods, Richard H. Overholt and Houck E. Bolton,
Brookline, Massachusetts.
Discussor: Henry A. Brodkin, Newark, New Jersey.

12:00 Noon—*Round Table Luncheon Meetings*

FRIDAY, JUNE EIGHTH (Continued)

2:00 p.m.—*Scientific Session No. 3*

Otto C. Brantigan, Baltimore, Maryland, Chairman,
William P. Chester, Detroit, Michigan, Secretary.

"Pneumothorax, When Should an Adequate Collapse be Expanded," (Committee Report)

Report of a survey made by the Committee on Non-Surgical Collapse Therapy, Council on Management and Treatment of Diseases of the Chest, American College of Chest Physicians.

Harold G. Trimble, J. Lloyd Eaton and Ina Gourley, Oakland, California.

Committee:

Harold G. Trimble, Oakland, California, Chairman
Duane Carr, Memphis, Tennessee, Vice-Chairman
A. Worth Hobby, Atlanta, Georgia, Secretary
John S. Packard, Allenwood, Pennsylvania
Harry Shubin, Philadelphia, Pennsylvania
Buford H. Wardrip, San Jose, California
Giles Wolverton, Dayton, Ohio
David Ulmar, New York, New York.

"X-Ray Surveys and Bronchogenic Carcinoma," (Committee Report)

Report of a survey made by the Committee on Surgical Treatment of Diseases of the Chest, Council on Management and Treatment of Diseases of the Chest, American College of Chest Physicians.

Alfred Goldman, Los Angeles, California.

Committee:

Alfred Goldman, Los Angeles, California, Chairman
Irving Sarot, New York, New York, Vice-Chairman
John Roberts Phillips, Houston, Texas, Secretary
Charles P. Bailey, Philadelphia, Pennsylvania
Karl P. Klassen, Columbus, Ohio
W. L. Rogers, San Francisco, California
Samuel A. Thompson, New York, New York
David H. Waterman, Knoxville, Tennessee.

"The Recognition of Nonallergic Asthma,"
Leon Unger, Chicago, Illinois.
Discussor: Harry H. Epstein, Jamaica, New York.

Symposium

"ACTH and Cortisone in Diseases of the Chest,"
Edwin R. Levine, Chicago, Illinois, Moderator.

Panel:

James M. Carlisle, Rahway, New Jersey
Smith Freeman, Chicago, Illinois
Laurance W. Kinsell, San Francisco, California
John R. Mote, Chicago, Illinois
Maurice S. Segal, Boston, Massachusetts.

Discussion between the audience and the panel members is invited.

8:00 p.m.—*Motion Picture Session on Diseases of the Chest*

Paul H. Holinger, Chicago, Illinois, Chairman.

"Pulmonary Hernia" (16 mm, color, silent, 10 minutes)
Lawrence M. Shefts, San Antonio, Texas.

"Conversion of the Auricular Appendage into a Leak-Proof Valve Tube for Intracardiac Surgery" (16 mm, color, silent, 16 minutes)
Samuel A. Thompson, New York, New York.

FRIDAY, JUNE EIGHTH (Continued)

"Prone Position in Thoracic Surgery (With Emphasis on Prevention of Operative and Post-Operative Complications)," (16 mm, color, sound, 18 minutes).
Emil A. Naclerio, New York, New York.

"Physiologic Therapy in Bronchial Asthma," (16 mm, silent, 12 minutes).
Alvan L. Barach, New York, New York.

"Spontaneous Rupture of the Esophagus," (16 mm, color, silent, 22 minutes).
S. Allen Mackler, Chicago, Illinois.

"Tuberculosis of the Larynx," (16 mm, color, silent, 18 minutes)
William A. Lell, Philadelphia, Pennsylvania.

"Endoscopic Appearances of Diseases of the Trachea," (16 mm, color, silent, 20 minutes).
Paul H. Holinger and Kenneth C. Johnston, Chicago, Illinois.

Saturday, June Ninth

10:00 a.m.—*Scientific Session No. 4*
David W. Heusinkveld, Cincinnati, Ohio, Chairman,
Orin J. Farness, Tucson, Arizona, Secretary.

"Studies in Pericarditis Cases,"
George R. Herrmann, Ernesto J. Marchand and Milton R. Hejtmancik, Galveston, Texas.
Discussor: George C. Griffith, Pasadena, California.

"The Clinical Syndrome of Truncus Arteriosus and Pseudo-Truncus Arteriosus,"
Helen B. Taussig and Albert Cohen, Baltimore, Maryland.
Discussor: Nathaniel E. Reich, Brooklyn, New York.

"Neurohemodynamic Pulmonary Edema: Autonomic Influence on Pulmonary Capillary Pressure and the Acute Pulmonary Edema State,"
Stanley J. Sarnoff, Boston, Massachusetts.
Discussor: Irving Mack, Chicago, Illinois.

"The Relationship Between Pathological Changes in Blood Vessels in Resected Lobes and Lungs as Correlated with Pulmonary Artery Pressure Changes Recorded During Cardiac Catheterization,"
Allan Hurst, Sidney H. Dressler and John Denst, Denver, Colorado.
Discussor: Herbert C. Maier, New York, New York.

12:00 Noon—*Round Table Luncheon Meetings*

2:00 p.m.—*Scientific Session No. 5*
Hugh G. Whitehead, Baltimore, Maryland, Chairman,
Clyde W. George, Buffalo, New York, Secretary.

"Exploratory Surgery of the Heart,"
Charles P. Bailey, Robert P. Glover and Thomas J. O'Neill, Philadelphia, Pennsylvania.
Discussor: D. W. Gordon Murray, Toronto, Canada.

"Severe Pulmonary Hypertension in Patent Ductus Arteriosus. Its Behavior After Ligature of the Ductus,"
Ignacio Chavez, S. Novelo R. Limon and E. Cabron, Mexico City, Mexico.
Discussor: Milton W. Anderson, Rochester, Minnesota.

3:00 p.m.—*X-Ray Seminar*
Leo G. Rigler, Minneapolis, Minnesota, Chairman.

Sunday, June Tenth

9:00 a.m.—*Scientific Session No. 6*

E. C. Drash, Charlottesville, Virginia, Chairman,
Alexander Libow, Miami Beach, Florida, Secretary.

"The Status of Tried Procedures in the Treatment of Pulmonary Tuberculosis as Seen in the Light of Thirty Years' Experience,"
William A. Hudson and Willard B. Howes, Detroit, Michigan.
Discussor: Henry W. Leetch, Saranac Lake, New York.

"Venous Pressure as a Guide to Pneumoperitoneum Therapy in Pulmonary Emphysema,"
Gustav J. Beck and A. Chesmore Eastlake, New York, New York.
Discussor: Leon H. Hirsh, Milwaukee, Wisconsin.

Symposium

"Chemotherapy and Antibiotics in Diseases of the Chest,"
Karl H. Pfuetze, Chicago, Illinois, Moderator.

Panel:

Alvan L. Barach, New York, New York
Sumner Cohen, Oak Terrace, Minnesota
Gladys L. Hobby, Brooklyn, New York
Alvis E. Greer, Houston, Texas
Eugene T. McEnery, Chicago, Illinois
Leopoldo Molinari, Lime, Peru
Carl W. Tempel, Denver, Colorado
Selman A. Waksman, New Brunswick, New Jersey.

Discussion between the audience and the panel members is invited.

12:00 Noon—*Round Table Luncheon Meetings*

2:00 p.m.—*Scientific Session No. 7*

Samuel A. Thompson, New York, New York, Chairman,
Ethan B. Pfefferkorn, Oshkosh, Wisconsin, Secretary.

"Recovery of Pulmonary Function After Thoracic Injury,"
Nathan Kenneth Jensen, Minneapolis, Minnesota.
Discussor: William M. Lees, Chicago, Illinois.

"Current Concepts on the Etiology of the Common Cold,"
Thomas G. Ward, Baltimore, Maryland.
Discussor: Alphonse R. Dochez, New York, New York.

Symposium

"Occupational Diseases of the Chest,"
Presented jointly by the American Association of Industrial Physicians and Surgeons and the American College of Chest Physicians.

Edward C. Holmblad, Chicago, Illinois, Moderator.

"Anthracosilicosis,"
Louis L. Friedman, Birmingham, Alabama.

"Silicosis"
Lloyd E. Hamlin, Chicago, Illinois.

"Pulmonary Function Tests"
George R. Meneely, Nashville, Tennessee.

"Treatment and Rehabilitation"
Frank Princi, Cincinnati, Ohio.

SUNDAY, JUNE TENTH (Continued)

"Classification of Dust and Siderosis"
Oscar A. Sander, Milwaukee, Wisconsin.

Panel:

Frank R. Ferlaino, New York, New York
Burgess L. Gordon, Philadelphia, Pennsylvania
Donald O. Hamblin, New York, New York
Edgar Mayer, New York, New York
Leo G. Rigler, Minneapolis, Minnesota
Reginald Smart, Los Angeles, California
Arthur J. Vorwald, Saranac Lake, New York
C. Richard Walmer, Pittsburgh, Pennsylvania.

Discussion between the audience and the panel members is invited.

Round Table Luncheon Meetings

"The Use of Artificial Pneumoperitoneum,"
James S. Edlin, New York, New York and
Louis L. Friedman, Birmingham, Alabama.

"Should the Custom of Doing Biopsies in Suspicious Tumors be Abolished?"
Paul H. Holinger, Chicago, Illinois; Karl P. Klassen, Columbus, Ohio and George G. Ornstein, New York, New York.

"Cor Pulmonale Associated with Chronic Pulmonary Disease,"
Milton W. Anderson, Rochester, Minnesota and
Irving Mack, Chicago, Illinois.

"Fungus Diseases,"
Hyman E. Bass, New York, New York; Alfred Goldman, St. Louis, Missouri and Alvis E. Greer, Houston, Texas.

"Recognition and Management of Chest Diseases in the Older Age Group,"
Benson Bloom, Tucson, Arizona; Burgess L. Gordon, Philadelphia, Pennsylvania and William B. Tucker, Minneapolis, Minnesota.

"Management of Emphysema,"
Hurley L. Motley, Philadelphia, Pennsylvania and
Herbert C. Sweet, St. Louis, Missouri.

"Management of the Severe Asthmatic,"
Maurice Segal, Boston, Massachusetts; Leon Unger, Chicago, Illinois and Orval Withers, Kansas City, Missouri.

"Indications and Results of Pulmonary Resection,"
Richard H. Overholt, Brookline, Massachusetts and
Eli H. Rubin, New York, New York.

"BCG Vaccination,"
Konrad Birkhaug, Albany, New York and
Milton I. Levine, New York, New York.

"Suppurative Diseases of the Lungs,"
Alfred Goldman, Los Angeles, California and
Henry C. Sweany, Jacksonville, Florida.

"Management of Cough,"
Andrew L. Banyai, Milwaukee, Wisconsin.

"Accuracy of Diagnostic Procedures in Bronchogenic Carcinoma,"
Seymour M. Farber, San Francisco, California; Peter A. Herbut, Philadelphia, Pa. and Arthur Q. Penta, Schenectady, New York.

"Management of Chest Diseases in Children,"
Lloyd B. Dickey, San Francisco, California; Eugene T. McEnery, Chicago, Illinois and Teodosio Valledor, Havana, Cuba.

"Allergy of the Respiratory Tract,"
George Piness, Los Angeles, California; Bret Ratner, New York, New York and Fred W. Wittich, Minneapolis, Minnesota.

ROUND TABLE LUNCHEON MEETINGS (Continued)

"Coronary Circulation,"
Ignacio Chavez, Mexico City, Mexico and
Harry Gold, New York, New York.

"Bronchitis and Bronchiectasis,"
Buford Wardrip, San Jose, California and
David H. Waterman, Knoxville, Tennessee.

"Management of Congestive Heart Failure,"
John F. Briggs, St. Paul, Minnesota and
John J. Sampson, San Francisco, California.

"Evaluation of Pulmonary Function,"
Earle B. Kay, Cleveland, Ohio; Peter A. Theodos, Philadelphia, Pennsylvania and George W. Wright, Saranac Lake, New York.

SATURDAY, JUNE NINTH:

6:00 p.m.—*Annual Convocation*
Louis Mark, Columbus, Ohio, President, presiding.
Report of the College Historian
William A. Hudson, Detroit, Michigan.
Awarding of Fellowship Certificates
James H. Stygall, Indianapolis, Indiana, Chairman, Board of Regents.

7:00 p.m.—*Cocktail Party*

7:30 p.m.—*Annual Presidents' Banquet*
Louis Mark, Columbus, Ohio, President, presiding.
Introduction of Guests.
Introduction of President-Elect
Chevalier L. Jackson, Philadelphia, Pennsylvania.
Presentation of College Medal.
Presentation of College Essay Award.

The College Medal will be presented to a prominent scientist for meritorious achievement in the specialty of diseases of the chest.

A cash award of $250.00 will be presented to the winner of the essay contest sponsored by the American College of Chest Physicians. Certificates of honorable mention will be awarded for second and third place.

Ladies Activities

FRIDAY, JUNE EIGHTH:

Get-Acquainted Luncheon, Ambassador Hotel
Arrangements to attend the luncheon may be made at the Ladies Registration Desk.

SATURDAY, JUNE NINTH:

Annual Convocation, 6:00 p.m.

Cocktail Party, 7:00 p.m.

Annual Presidents' Banquet, 7:30 p.m.

Ladies Reception Committee:
Mrs. Irving Willner, Chairman
Mrs. Louis Mark, Honorary Chairman
Mrs. Lewis Baum
Mrs. S. Eugene Dalton
Mrs. Max Gross
Mrs. Charles Hyman
Mrs. Herman Miller
Mrs. Benjamin P. Potter
Mrs. Albert D. Tucker.

College Chapter News

ILLINOIS CHAPTER

A joint meeting of the Illinois Chapter of the College and the Chicago Tuberculosis Society was held at the Congress Hotel, Chicago, on Friday, April 20. A panel discussion on the subject of the heart in relation to pulmonary diseases was presented in which the following physicians participated:

Louis N. Katz, Chicago, Illinois, Moderator
John F. Briggs, St. Paul, Minnesota
Egbert H. Fell, Chicago, Illinois
Aldo A. Luisada, Chicago, Illinois
Chauncey C. Maher, Chicago, Illinois.

Abel Froman, Chairman, Program Committee, Illinois Chapter.

OHIO CHAPTER

The Ohio Chapter of the College met at the Netherland Plaza Hotel, Cincinnati, on Wednesday, April 25. A luncheon and business meeting was held and the guest speaker, Dr. Harold I. Humphrey, Cincinnati, then spoke on the subject of "The Treatment of Tuberculosis with Thiosemicarbazone."

Harold G. Curtis, Secretary.

PHILIPPINE CHAPTER

The Philippine Chapter of the College held its annual meeting on March 27 at which time the following officers were elected:

Carmelo P. Jacinto, Manila, President
Manuel Quisumbing Sr., San Pablo, Vice-President
Laureano Bautista, Quezon City, Secretary-Treasurer.

Professor Gustav Maurer, Davos, Switzerland, and Dr. Maurice Gilbert, Geneva, Switzerland, have been appointed observers at the Fourth World Health Assembly to be held in Geneva on May 7. Professor Maurer and Dr. Gilbert will officially represent the American College of Chest Physicians at this meeting. The College was recently made a member organization of the World Health Organization.

Dr. Frank R. Ferlaino, New York, New York, has been appointed a member of the Board of Trustees of the Society of Medical Jurisprudence of New York State.

Dr. Edward Dunner has been appointed VA area section chief of tuberculosis for the eighth south-central and midwestern states with headquarters in St. Louis, Missouri. Dr. Dunner was formerly chief of the tuberculosis service of the Veterans Hospital at Livermore, California.

Dr. Daniel E. Jenkins, Houston, will present a paper on "Recent Trends in the Treatment of Tuberculosis" in the Section on Internal Medicine of the State Medical Association of Texas, meeting in Galveston, May 1 and 2. Dr. David McCullough, Kerrville, will officially open the discussion of Dr. Jenkins' paper.

COLLEGE EVENTS

17th Annual Meeting, American College of Chest Physicians, Atlantic City, New Jersey, June 7-10, 1951.

New York State Chapter Meeting, Buffalo, May 3, 1951.

California Chapter Meeting, Los Angeles, May 12, 1951.

Illinois Chapter Meeting, Chicago, May 23, 1951.

Potomac Chapter Meeting, Washington, D. C., May 24, 1951.

Chicago Postgraduate Course, September 24-28, 1951.

Minneapolis Postgraduate Course, October 18-20, 1951.

New York City Postgraduate Course, November 12-17, 1951.

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Physician-in-charge of medical care wanted for university hospital. Starting salary $600 monthly. For further information please address Box 219A, American College of Chest Physicians, 500 N. Dearborn St., Chicago 10, Ill.

Residency in thoracic surgery available for well qualified man at university hospital. Please address Box 220A, American College of Chest Physicians, 500 N. Dearborn St., Chicago 10, Ill.

Resident for 175-bed, fully approved, tuberculosis hospital; salary $3,000.00 per year, less nominal sum for maintenance; to start June 1, 1951. Please apply to Medical Director, Mahoning Tuberculosis Sanatorium, 4880 Kirk Road, Youngstown 7, Ohio.

Staff physician wanted for 160-bed tuberculosis hospital situated in the Pacific Northwest. Physician should be eligible for Oregon license. Salary $495 to $555 with complete maintenance. Full particulars given upon request. Please address all inquiries to Box 221A, American College of Chest Physicians, 500 N. Dearborn St., Chicago 10, Illinois.

Two thoracic surgeons offer attractive position to either a medical or surgical associate. Practice confined to diseases of the chest. Young, well-trained, middle-westerner, or westerner preferred. Board qualification desirable. For further particulars, please address Box 222A, American College of Chest Physicians, 500 N. Dearborn St., Chicago 10, Illinois.

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Well trained Chinese thoracic surgeon desires surgical or medical-surgical residency in sanatorium or hospital for chest diseases. Graduate of Grade A medical school in China, 1943. Fellow ACCP. Wife trained in nursing and as laboratory technician. For further information please address Box 254B, American College of Chest Physicians, 500 N. Dearborn St., Chicago 10, Illinois.

NEW BCG LABORATORY AT JAFFA

The foundation stone for a new BCG laboratory has been laid at the former Dajani Hospital at Jaffa, Israel. Within a period of seven months Israel will produce her own BCG vaccine. While about a third of the country's population has been examined and most of them inoculated against tuberculosis in the International Tuberculosis Campaign, the goal is the immunization of all persons under 18 years of age.

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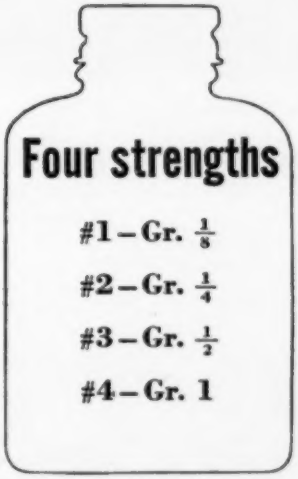

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